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PART I., 1895.

Original Articles and Clinical Cases.

EXPERIMENTAL ENQUIRY UPON THE AFFERENT TRACTS OF THE CENTRAL NERVOUS SYSTEM OF THE MONKEY.

BY F. W. MOTT, M.D.

Lecturer on Physiology at Charing Cross Hospital.

Introduction.—Since the adoption of the Marchi method for tracing the course of degenerating fibres in the central nervous system, many results have been obtained which were not capable of demonstration by the previous methods of Weigert and Pal. We can now follow single degenerated fibres in their course, and provided all sources of fallacy, such as mistaking every black dot for a degenerated fibre, be avoided, the method is a most valuable one; but in order to ensure correct results with such a very sensitive method, no pains must be spared to ensure the elimination of all possible sources of fallacy. From a long experience, I have concluded that in order to distinguish between black particles and degenerated fibres in transverse section, it is essential so to cut the sections that in some part of their course these degenerated fibres can be seen in vertical section, or in sections which display *a length* of the degenerated nerve fibres. Since probably every degenerated fibre will show itself, great care must be taken in the operative procedure; firstly, the wound must be and remain perfectly aseptic; secondly, the antiseptic precautions used must be of a non-irritating character (I always use boiled saline solution); thirdly, the lesion must be limited to the structures which are to be experimented upon, for in opening the spinal canal, especially of small animals, it is extremely easy to damage roots which should be left intact, and to compress the cord; lastly, in cutting roots it must be remembered how very easy it is in certain situations, even with the greatest care to injure the cord, and that in dividing roots unilaterally, especially when a larger number are cut, it is often unavoidable not to interfere with the vascular supply of the posterior part of that half of the spinal cord. Again, there seems to be some evidence to show that certain fibres degenerate when cut off from their trophic centre *at different rates*. By considering these possible fallacies many degenerations which have lately been described may probably be explained away. The experiments will be found to fall into three groups, A, B, and C, as below.

A.—*Summary* of results of experiments upon unilateral section of roots of the lumbo-sacral region of the spinal cord in seven monkeys and one cat.

Degenerated fibres do not pass into the opposite posterior column, nor do they probably enter directly into any of the *long* ascending tracts of the anterior or lateral columns. In the upper part of the cord the degeneration is limited absolutely to the posterior median column of the same side. These facts are proved by three successful cases in which presumably none of the possible fallacies occurred; therefore the degeneration was strictly limited to the part mentioned. These were Cases 1, 2, 3 (*vide infra*). The degeneration that was found in four cases, and which made me believe at first that fibres passed directly into the opposite antero-lateral tract may probably be explained by one of the fallacies mentioned. In Cases 5, 6 and 7 there was degeneration in the opposite posterior column readily explicable by accidental causes.

Goll's column is formed principally by fibres from the fifth, sixth and seventh post-thoracic roots, but some fibres forming a small triangle posteriorly come presumably from the tail.

I am indebted to my friend Dr. Sherrington, with whom I am now carrying on further observations on this subject for material of some of the subjoined cases. (*Vide* fig. 1 and plate I., photo-micrographs 1, 2, 3, 4.)

EXPERIMENT I.—Unilateral section of all the posterior roots below the third lumbar on the right side.

The animal was kept alive for two months, and the spinal cord hardened in Müller's fluid, and sections stained by Marchi fluid were cut in celloidin. The following degenerations were noticed:—The whole posterior column of the same side degenerated as high as the second lumbar, then healthy fibres began to appear in the postero-external column. There was *no* degeneration in the opposite posterior column nor in the lateral columns above the lesion, nor in the cross pyramidal tracts below the third lumbar.

EXPERIMENT II.—Section of the large sixth sub-thoracic posterior root on the right side, below the level of the spinal cord.

This is a very important root, as the degeneration shows in the formation of the posterior column in the lumbo-sacral region, and its fibres enter largely into the formation of Goll's column. The animal was kept alive a fortnight; absolutely no degeneration was observed in any other part of the cord above the lesion than in the posterior column of the same side. (Photo-micrograph, plate I., 2.)

EXPERIMENT III.—Cat. Unilateral section of the posterior spinal ganglia, fifth, sixth, seventh, eighth and ninth post-thoracic roots.

No degeneration except in the posterior column of the same side. Animal kept alive forty days.

EXPERIMENT IV.—Unilateral section of fifth, sixth, and seventh post-thoracic roots. Animal kept alive ten days.

Degeneration in opposite posterior column, and in cross-pyramidal tracts below lesion and in lateral columns above lesion a scattered degeneration. This was probably due, as results show, to pressure on the cord during the operation, or subsequently from inflammatory products.

EXPERIMENT V.—Unilateral section of the third, fourth, fifth, sixth, seventh, eighth and ninth sub-thoracic in a monkey.

Animal kept alive one month. Degeneration limited to the posterior column of same side above lesion. (Micro-photograph 1, plate I.) No degeneration in opposite posterior column. About thirty degenerated fibres in the situation of Gower's tract in the opposite lateral column. (See plate I.) At first I thought that the only explanation of the presence of these fibres was that posterior root fibres pass directly through the anterior commissure to the opposite antero-lateral column, but their absence in Cases 1, 2, and 3 leads me to suspect *that some cells in the grey matter* have undergone degenerative changes, and consequently fibres which arise from these cells have degenerated upwards and can be traced as far as the corpora quadrigemina. In Experiment I. a larger number of roots were divided, and yet there was no trace of this tract of degeneration. We are, therefore, not warranted in assuming more than that impulses do pass from the posterior roots of the right side to the left corpora quadrigemina by these thirty-odd fibres, which we find degenerated as a result of the operation. Possibly thrombosis of a vessel running up the posterior roots and supplying a group of nerve cells in connection with these posterior roots below and with the antero-lateral fibres of the opposite side above may be the explanation.

EXPERIMENT VI.—Unilateral section on the right side of third, fourth, fifth, sixth, and seventh posterior roots in a monkey.

Animal kept alive eighteen days. Degeneration in both antero-lateral columns above lesion and in posterior column of opposite side. This was caused by injury of the sixth sub-thoracic root on the left side in opening the spinal canal. (Plate I., photo-micrograph 3).

EXPERIMENT VII.—Unilateral section of the fourth post-thoracic nerve, outside the dura mater. Animal kept alive ten days.

I found very little degeneration in the column of Goll of the same side in the cervical region.

EXPERIMENT VIII.—Unilateral section of the fourth, fifth, sixth, seventh, eighth and ninth sub-thoracic roots, Animal kept alive two months. Scattered degenerated fibres in both antero-lateral columns above the lesion, a very few fibres in the opposite posterior column. The degenerated fibres in the opposite antero-lateral tract were about thirty in number, and could be traced as far as the corpora quadrigemina.

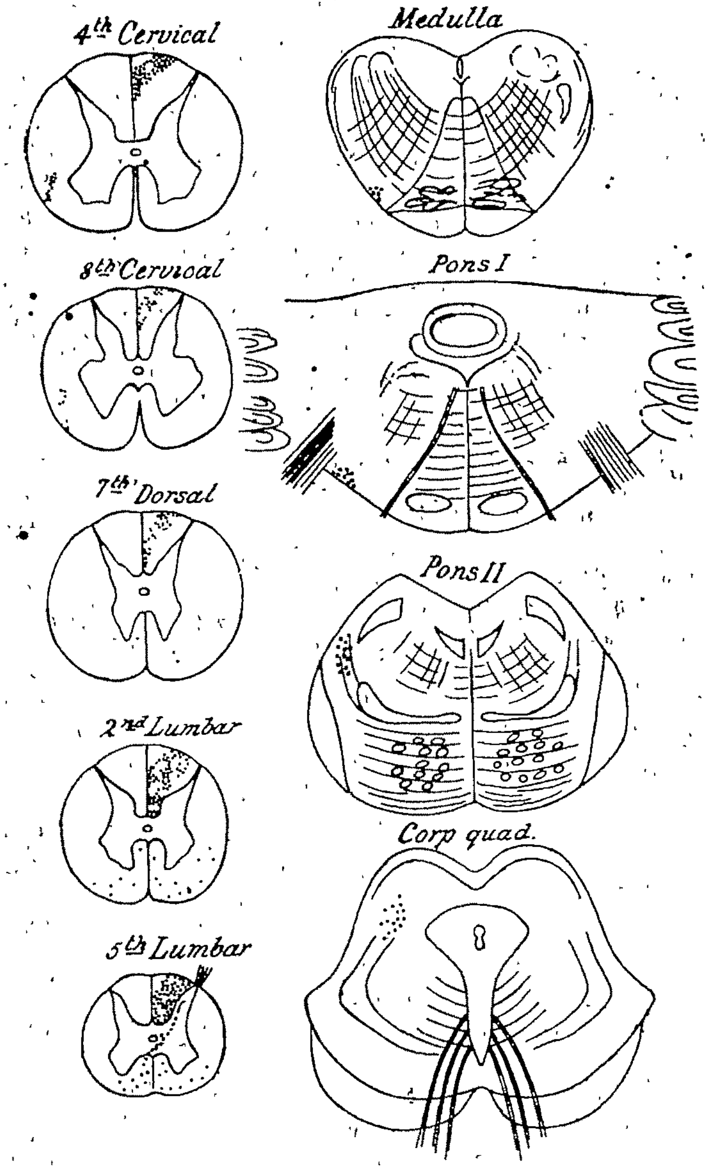

FIG. 1.

Representation of degeneration resulting from unilateral section of a number of roots entering into formation of lumbo-sacral plexus (F. W. M.). The antero-lateral degeneration does not always occur, and it is concluded that when it does it is probably due to vascular changes induced in the grey matter by section of vessels contained in the posterior roots divided. The number of degenerated fibres does not bear a proportion to the number of roots divided, and are not always present when a large number are divided. It obviously corresponds in position and destination with the innermost tract of crossed ascending fibres of fig. 2. The small triangular area of undegenerated fibres seen posteriorly is represented larger than it should be to make it obvious, and I think it represents fibres from coccygeal roots. It varies in size in different animals.

With regard to the results of these experiments I feel convinced that fibres do not pass into the opposite posterior columns from the roots which I have cut in these eight cases. When fibres are found degenerated in the crossed afferent tract of Gowers and Edinger, I would rather explain the degeneration by vascular changes in the grey matter of the posterior horn, than by supposing that fibres from the posterior roots pass directly across to ascend the cord and reach the corpora quadrigemina. On the whole the results of my experiments do not confirm the results of Lowenthal, Berdez, Oddi and Rossi and others, who have found extensive degeneration elsewhere than the posterior column of the same side. These experiments rather confirm the results of my previous experiments on monkeys. I found, moreover, that if all the nerve roots entering into the formation of the lumbo sacral plexus, be divided from the third downwards, just above the uppermost cut root, the whole of the posterior column of the same side will be degenerated (*vide* photo-micrograph 1, plate. i.), but as soon as the fibres of an uncut nerve enter they occupy the part of the column nearest the grey matter. While one group of the degenerated fibres passes forwards and upwards into Clarke's column, ending in a brushwork around the vesicular cells, the other group of fibres passes medianwards and backwards to reach the column of Goll, in which they ascend vertically, and the fibres that enter by the lowest roots take up apparently the most posterior position.

B.—*Summary* of results of median section of the spinal cord in the lumbar region in two monkeys (fig. 2).

B. EXPERIMENT I.—Median section of the spinal cord for ¾ in., involving the last dorsal and upper three lumbar-segments.

EXPERIMENT II.—Median section for about ¾ in., involving third, fourth and fifth lumbar segments.

The operations were performed with strict antiseptic precautions, and all the while the animals lived no evidence of hyperæsthesia was observed. In both cases, however, the lower limbs were considerably enfeebled, both in sensibility and movement. In Case 1, since the section was not quite in the mid-line, the gray matter of one side was considerably damaged and the

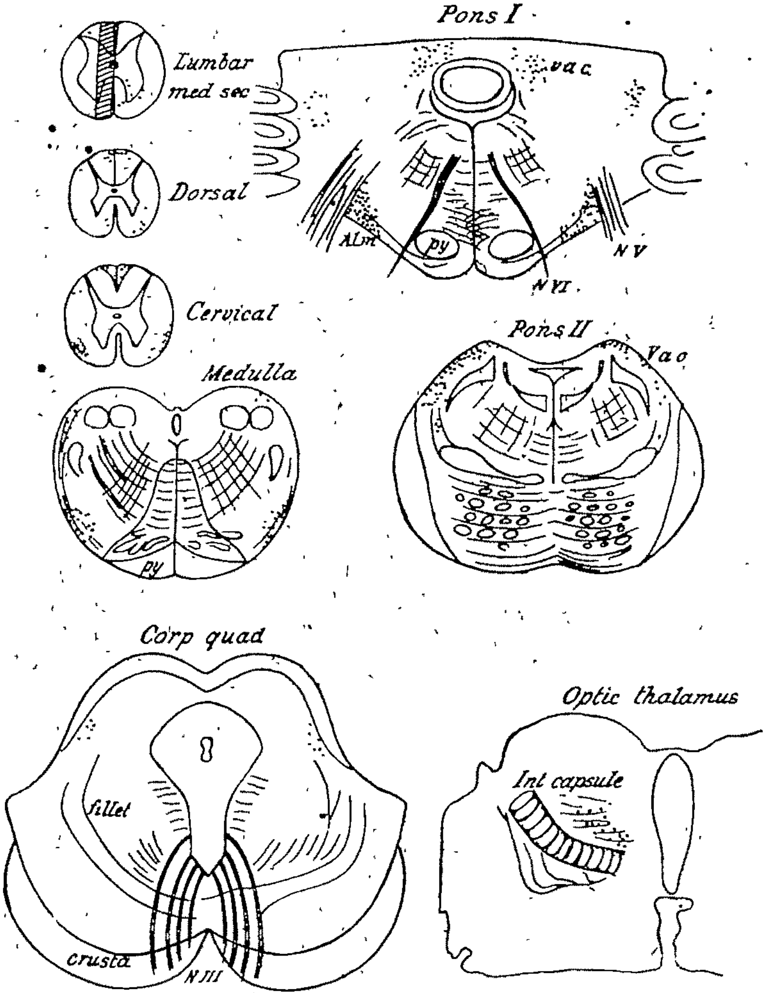

Fig. 2.

Degenerations resulting from median longitudinal section of the spinal cord in the lumbar region (F. W. M.). Symmetrical ascending degeneration in the antero-lateral ascending tracts. One tract (ventral cerebellar) goes to the vermis vac., the other to corpora quadrigemina.

limb of the corresponding side was more paralysed and certainly less sensitive. It was, moreover, swollen, probably owing to vaso-motor disturbance. I adopted the same methods for examining, microscopically, the results, as in the previous related experiments.

In Experiment I., the lesion was not in the mid-line, but involved the anterior and posterior horn of the left side, not injuring the grey matter of the right side at all.

There was symmetrical degeneration in the antero-lateral column of both sides, not so, however, of the direct cerebellar tract, which was much more extensively degenerated on the left side—the side of injury of the grey matter. This lesion was higher than the next, involving the last dorsal segment, and therefore the lower end of the posterior vesicular column of Clarke. (*Vide* photo-micrograph 4, plate I.).

In Experiment II., the lesion was much more in the mid-line and was situated below the position of Clarke's column.

There was very little degeneration of the direct cerebellar tract, but symmetrical degeneration of the antero-lateral ascending tracts. On examining sections of these spinal cords in the cervical region (see fig. 2), it can be observed that there are two distinct sets of fibres degenerated, one (the more extensive) occupying especially the periphery, *ventral cerebellar*, the other much smaller stretching backwards and inwards, separated from the former by healthy fibres. This latter tract corresponds in position to the tract of fibres, observed in the opposite antero-lateral column in some of the cases of unilateral section of posterior roots, except that the number of degenerated fibres is much more numerous. The ventral cerebellar tract in the monkey, as I have already shown,* can be traced to the middle lobe of the cerebellum forming a loop over the fifth nerve and turning downwards and backwards on the posterior surface of the superior cerebellar peduncle to reach its destination. The other set of ascending fibres could be traced upwards as far as the superior corpora quadrigemina lying outside the fibres of the lateral fillet, a few *apparently* going on with the fibres of the fillet to the optic thalamus. This is in all probability the crossed sensory tract of Edinger.

C. *Summary of Experiments of Unilateral Separation of the Nuclei of the Posterior Columns from the Arciform Fibres issuing therefrom.*—Five experiments on monkeys, which

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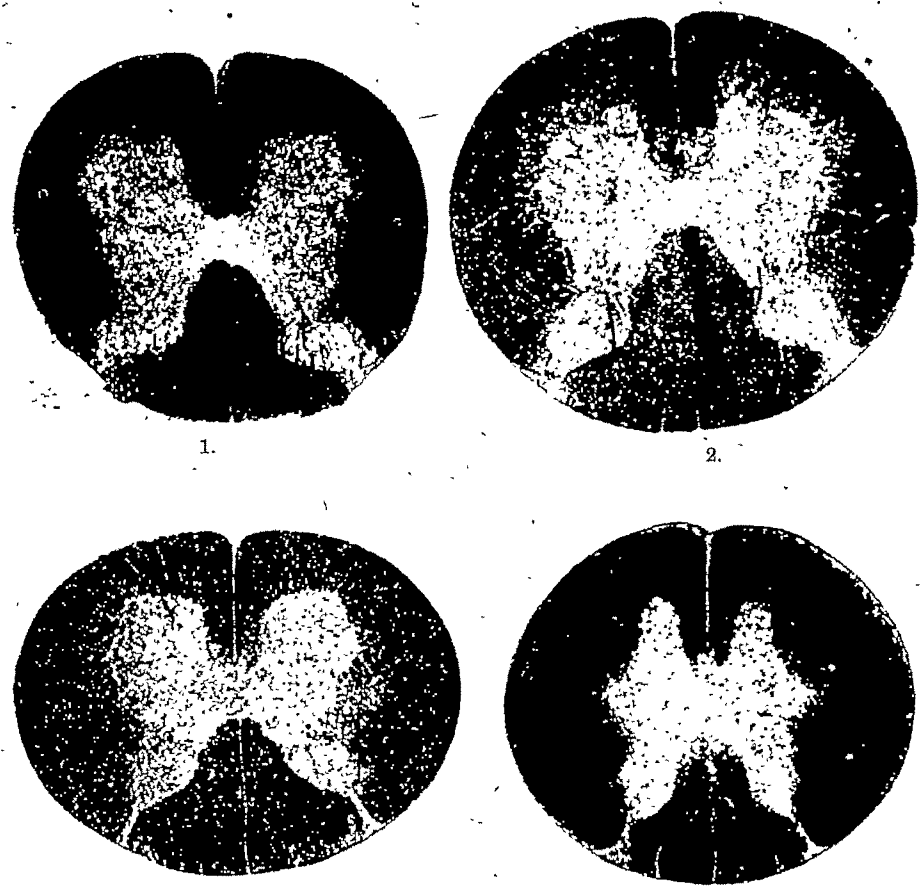
* Ref. 5.

PLATE I.

DEGENERATIONS FROM UNILATERAL SECTION OF POSTERIOR ROOTS.



1. 2.

3. 4.

Unilateral section of posterior roots in the monkey. (1) Spinal cord at level of third lumbar after unilateral section of posterior roots of cauda equina, experiment v. Degeneration only in posterior column of same side. (2) Section of the sixth subthoracic root (first sacral man.) Degeneration of posterior column in the upper part of lumbar enlargement, experiment ii. (3) Degeneration of Goll's column; cervical enlargements after unilateral section of posterior roots of cauda equina. (4) The degeneration found in the upper dorsal region after median section. Exp. I.B. The lesion was not quite in the mid-line, and involved the lower end of Clarke's column. There is in consequence degeneration of the direct cerebellar tract.

F. W. MOTT.

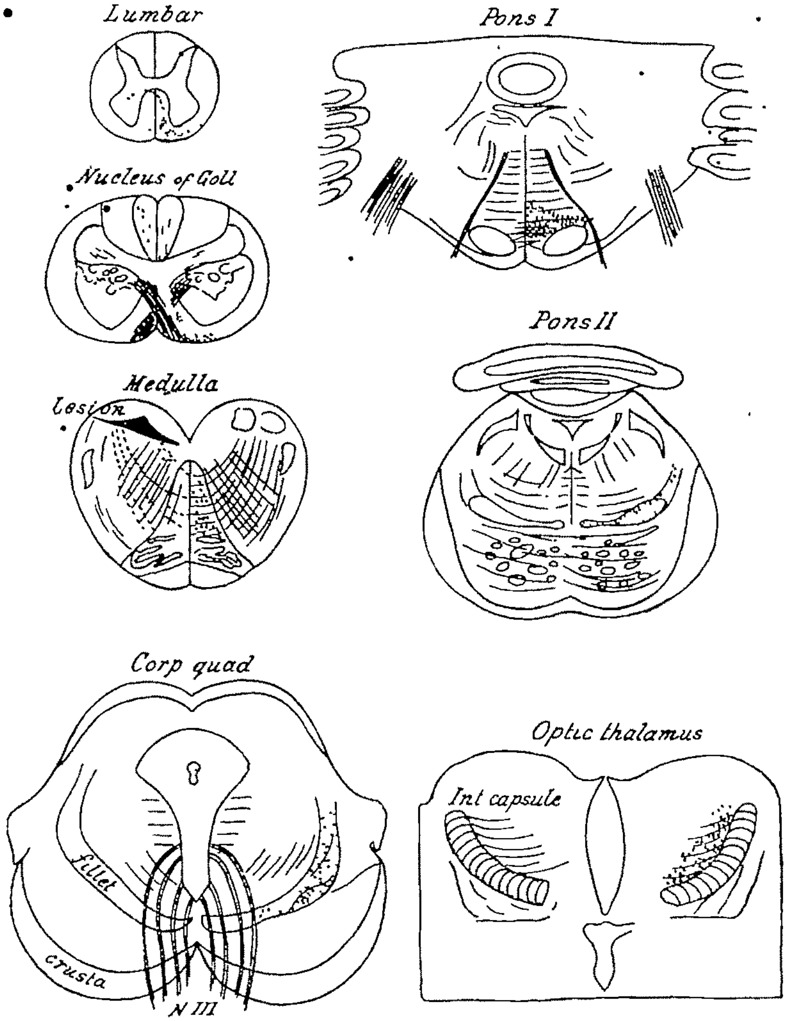


FIG. 3.

Degenerations in the internal arciform fibres, interolivary layer and of the fillet of the opposite side, following unilateral separation of the posterior column nuclei (F. W. M.). The degenerated fibres represented by black dots can be followed as high as the optic thalamus when they end amid the skin medullares. In some cases of extensive lesions there was a degeneration down the cord probably due to injury of ground fibres. This degeneration is represented in the antero-lateral column of the lumbar region; some are seen decussating in the anterior commissure.

produced the following results of degeneration illustrated by fig. 3, plates II. and II. A, viz., degeneration of the arciform fibres issuing from Goll's nucleus and Burdach's nucleus, which decussate across the mid-line to the opposite inter-olivary layer, forming in the upper part of the medulla a triangular area of degeneration. In the upper part of the pons the degenerated fibres are seen in the median and lateral fillet; probably some of the fibres in the lateral fillet terminate in the corp. quad., while others (more numerous) especially those of the median portion, can be traced onwards to the *optic thaalmus*, where they spread out and terminate in that mass of grey matter passing through the *striæ medullares.* In not one of these five cases could I satisfy myself that there was a direct continuity of these degenerated fibres *by a cortical fillet* with the cortex cerebri. In fact, they all seemed to terminate in the optic thalamus. In two cases the injury extended rather deeper and higher, involving the ground fibres and some of the cranial nuclei situated in the floor of the fourth ventricle, and a considerable degeneration was noted in consequence in the spinal cord *below the lesion* in the anterior columns, extending down to the lumbo-sacral region. Most of these fibres *decussate in the anterior commissure.* (See fig. 3.) At first I thought that these might be due to injury of the restiform body, in accordance with the views of Marchi, but the researches of Ferrier and Turner, and Risien Russell[14] on the cerebellum appear to have entirely disproved Marchi's results.

I will not describe each experiment, as they were all conducted with one object, although sometimes more, sometimes less was done than intended. The following was the mode of procedure:—As in all the other cases of course the animal was *deeply anæsthetised*, and the part to be operated upon shaved and rendered *thoroughly aseptic.* An incision extending from the occipital protuberance down the neck in the mid-line for about one and a-half inches was made. The muscles attached to the occiput were separated from the bone until the occipito-atloid ligament was exposed. This was cut close to the bone all round. By keeping the neck well extended the floor of the fourth ventricle was exposed, so that one could see the lower part of the posterior surface of the medulla and the tip of the middle lobe of the cerebellum. A small, specially-constructed knife, bent at a suitable angle, was inserted into the lower part of the medulla to one side of the mid-line and thrust upwards so as just to cut away, unilaterally, the posterior column nuclei from their connections. *In all cases the lesion was unilateral* and was not complicated by any hæmorrhage or any difficulties. Nothing but *sterile saline solution* was used after exposing the occipito-atloid ligament—the muscles and the skin were sewed with horse-hair sutures and a collodion dressing applied. *The wounds healed invariably by first intention and remained aseptic.*

The physiological results were difficult to observe, but it seemed to me quite evident that in extensive lesions the animal did not use the hand of the same side with the same facility as on the opposite side. There was no pyramidal degeneration in most of these cases to account for this. With regard to sensation, it was quite impossible to say anything definite. In the notes of two of the cases I find that I remarked distinct lowering of sensibility on the side of the lesion. The degeneration which results from this lesion is exemplified in fig. 3, and in the photo-micrographs, plate II. and plate II. A. The brains and spinal cords of these five animals were hardened in Müller's fluid, stained by Marchi's method, and the whole of the central nervous system above the lesion, examined microscopically by serial sections, except the frontal lobes and the occipital lobes. This represents, of course, an immense amount of labour and the preparation of a great number of sections.*

COMPARISON OF RESULTS WITH PREVIOUS OBSERVATIONS AND EXPERIMENTS.

The experiments performed on the fibres of the posterior roots in monkeys confirm the results I obtained previously by the Weigert method, and agree with those of Singer[2] in the dog. I have made these experiments and observations in order to ascertain if Löwenthal,[3] Berdez,[4] and Oddi[4] and Rossi's experiments and observations are to be relied upon as showing that fibres pass directly into the long ascending tracts of the lateral columns and the opposite posterior column. Inasmuch as three of my cases failed to show such degeneration in the lateral columns, and four of my cases uncomplicated by injury of roots of the other side did *not show* any degenerated fibres in the opposite posterior column, I must conclude that probably some fallacy has existed. In fact, one of Löwenthal's cases is obviously fallacious, because there was considerable injury of the posterior horn. Löwenthal divided one or several posterior roots in various regions of the spinal cord in dogs, rabbits, and guinea-pigs. Stained by Marchi's method, his sections showed in the anterior commissure and in the anterior ground bundle, a degeneration which could be traced up the cord to the cervical region, gradually becoming less and less, and situated more at the periphery, also degenerated fibres of posterior column of same side and scattered degenerated fibres of the *opposite posterior columns*, but less numerous; degeneration of ventral cerebellar tracts of both sides more marked on the side of the lesion; and degeneration of intermedio-lateral tract on both sides. I cannot speak about sections of roots above the third lumbar, but I can certainly say that unilateral section of the large sixth sub-thoracic root—the unilateral excision of five ganglia in the cat and the section of the whole of the posterior roots entering into the formation of the lumbo-sacral and coccygeal plexuses on one side, produced no such degenerations—the only *long fibres* degenerated in these cases being the posterior column fibres of the same side.*

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* The sections from Experiments A, B, and C were shown at a meeting of the Physiological Society, held at Charing Cross Hospital Medical School, December, 1893, and at the Physiological Section of the British Association held at Oxford, 1894.

Oddi[4] and Rossi have described degenerated fibres in the opposite posterior column.

Berdez divided roots on one side in guinea-pigs. Some of the lesions injured the posterior horn and posterior columns, but Berdez concludes from his researches that fibres pass into both lateral columns and into the opposite posterior column.

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* Of course, it might be urged that the animal (Experiment I.) was kept alive too long to reveal the degeneration well, but that cannot be said of Experiment II. Again, that there are individual peculiarities in structure in different animals.

There is no doubt that these observers found degenerated fibres in the situations described, but I cannot help thinking that their presence can be explained by some such accidental cause as I mentioned in the commencement of my paper.

At first I believed that some few fibres did pass directly into the antero-lateral columns, *but as I could not find them in every case* I must conclude that these degenerated fibres come from cells in the grey matter. I think the results of median sections, as well as the presence of degenerated fibres on the opposite side in one case after a strictly unilateral root lesion, show that *Edinger*[5] and Gowers are right in supposing that *there is a path for afferent impressions from the posterior roots through cells of the grey matter, and by fibres from the latter which decussate in the anterior commissure* and arrive in the antero-lateral tracts, then ascend the cord and course in or near the lateral fillet, terminating in the corpora quadrigemina, and perhaps some few going into the thalamus. I cannot, however, agree with the assumption that this tract conveys painful impressions (Gowers). Its functions are not known. I have[6] divided the antero-lateral portions of the cord in the monkey on one and on both sides without any obvious effect on sensation. Lately Bechterew[7] has shown that section of the anterior half of the cord produces little or no effect on sensation in the dog. He finds that the only way to *produce* a complete block to sensory impulses is to cut through both lateral columns and the *posterior half of the cord as well.* Bottarzi[26] working with Luciani, has obtained similar results in dogs to those which I obtained in monkeys after hemisection of the spinal cord.

Brown-Séquard[8] himself has entirely renounced his view that the group of clinical symptoms which bears his illustrious name, and which results from unilateral stabs or wounds of the spinal cord (whether they be hemisections or not) can be explained by the immediate decussation of sensory impulses; indeed, just prior to his death he published a paper setting forth his later experiments and reasons why he had entirely renounced his former hypothesis.

We seem indeed to be coming back to the views of Schiff that the grey matter conducts painful sensations and heat and cold, whilst the posterior columns convey the tactile and muscular sense impressions. This is strongly supported by the observations made in recent years upon syringomyelia.

The fact I have above shown that that portion of the antero-lateral tract which can be traced to the vermis, and which is also *symmetrically* degenerated by median sections even when *the grey matter of one side* is seriously injured, the other side not, agrees with the results of Auerbach,[9] who found that destruction in the cat of a considerable length of one half of the spinal cord, including the posterior white column, the posterior horn of grey matter and the posterior part of the lateral column, is followed by ascending degeneration in the direct cerebellar tract of the same side and the antero-lateral cerebellar tract of the opposite, which he, like Lowenthal and myself, traces to the vermis.

In the median sections of the cord the antero-lateral fibres are seen to consist of two distinct tracts, one the crossed afferent tract of Edinger, the other a crossed cerebellar tract. I have shown that these tracts must arise from cells in the grey matter (perhaps at the base of the anterior horn), that they give off fibres which decussate in the anterior commissure; if they did not decussate then (like the direct cerebellar tract in the case in which the section was not in the mid-line, but involved the gray matter of one side), the degeneration of these two antero-lateral ascending tracts would not be equal and symmetrical on the two sides, but much more on the side in which the grey matter was destroyed. This inference is supported, moreover, by the experiments of Ehrlich and Brieger,[10] and Singer[11] and Münzer, viz., compression of the abdominal aorta in rabbits causes necrosis of the nerve cells in the lumbar spinal cord, and ascending degeneration in the antero-lateral ascending tract. It has been inferred by the latter two observers that this tract arises from the cells of the grey matter of the lumbar region. *Vide* also a paper by A. S. Grünbaum,[12] who confirms the observations of Auerbach and Singer and Münzer, but his observations are limited to the cord.

He considers as the result of his two experiments of longitudinal division of the spinal cord in the cat that the anterior commissure fibres go in part to the antero-lateral column, and at the same time derive their origin from the grey matter of the opposite side.

Schäfer[25] has figured fibres of the antero-lateral tract going to the corpora quadrigemina after hemisection of the spinal cord in the monkey.

THE POSTERIOR COLUMN NUCLEI AND THE FILLET.

A large and important literature exists upon the fillet based upon embryological and pathological data. For a full account, *vide* Flechsig and Hösel,[15] Mahaim,[16] Bruce,[17] Déjérine,[18] Von Monakow,[19] but as my paper is solely an experimental enquiry I shall only refer here especially to *experimental evidence* upon the origin and course of the fibres arising from the posterior column nuclei.

Von Vejas[20] discovered that after destroying the posterior column nuclei in new born animals there occurred an atrophy of the internal arciform fibres of the same side, atrophy of the inter-olivary layer of the opposite side, and atrophy of the fillet, which could be followed up as high as the corpus trapezoides. Lastly, Singer and Münzer,[21] after experimental destruction of the nuclei of Burdach found atrophy of the internal arciform fibres, and a demonstrable atrophy of the fillet of the opposite side which could be followed up to the optic thalamus.*

Monakow[19] found after destruction of the parietal lobe in young animals atrophy of the fillet, of the internal arciform fibres of the opposite side, and of the nucleus gracilis of the opposite side. This, certainly, taken with the observations of Flechsig and Hösel upon a case of porencephalic defect of the central convolutions which was associated with atrophy of the fillet of the same side and of the internal arciform fibres and the posterior column nuclei of the opposite side, shows that the fillet may degenerate downwards and that the posterior column nuclei are in direct connection (or indirect connection through the thalamus) with the central convolutions of the cortex.

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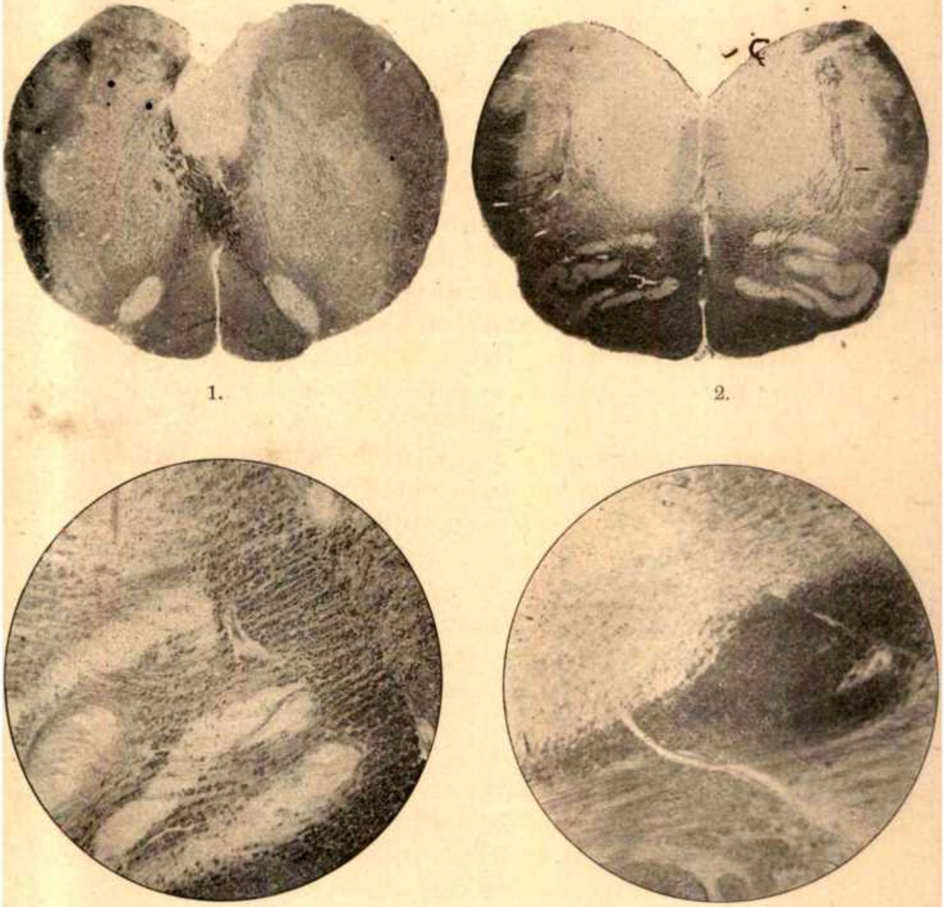
* I am informed by Dr. Turner that, in conjunction with Professor Ferrier their experiments showed that the fillet terminated in the optic thalamus.

As I was unable to trace any degeneration beyond the optic thalamus, or to find any evidence of a degenerated cortical fillet, I should be inclined to agree with Mahaim and Monakow that the posterior column nuclei stand in connection with the optic thalamus by the fillet, and each portion of the thalamus is in relation to a definite part of the cortex, especially as he, Monakow, finds that the degeneration may proceed down to the posterior column nuclei leading to their atrophy and destruction; may not, therefore, the cells in the optic thalamus undergo a similar atrophy and disappearance? The work of Flechsig and Hösel is, however, of very great importance, because it shows that an afferent tract carrying sensory impulses from the periphery is in connection with what is called the "motor area," and which, therefore, cannot be *purely motor*. The internal arciform fibres form two groups, one coming from Goll's nucleus and decussating at a lower level, the other coming from Burdach's nucleus. I am inclined to think, in contradistinction to Bechterew that the fibres proceeding from Goll's nucleus enter more into the formation of what is called the lateral fillet. I am of opinion that the distinction of different fillets is artificial, for it seems to me that the fibres of the so-called lateral fillet are fibres which are taking an oblique instead of a vertical direction. It is impossible to localise a lesion to Goll's nucleus, or to Burdach's nucleus alone, but I have formed this opinion from examination and comparison of sections in which these two nuclei have been unequally injured, and by other facts which, if confirmed by further observations, would render the supposition that fibres issuing from Goll's nucleus enter into the formation of the lateral fillet still more probable.

With regard to the degenerated fibres which descend the cord, many of which decussate in the anterior commissure, it is very possible, as suggested by Ferrier and Turner, that these come from Deiter's nucleus which is in relation with the semi-circular canals by the external branch

PLATE II.

POSTERIOR COLUMN NUCLEI LESIONS—PRODUCING DEGENERATION OF FILLET.



1. 2.

3. 4.

Lesions of medulla separating the posterior column nuclei from the arciform fibres issuing therefrom. (1) Lesion of nucleus gracilis showing the degenerated arciform fibres decussating to take up a position between the olive and pyramid. (2) Lesion higher up and separating Burdach's nucleus from the arciform fibres that issue from it. These degenerated fibres can be traced on the right hand side as the internal arciform fibres which sweep round behind the olive of the same side to reach the opposite interolivary layer. (3) Interolivary layer in the upper part of the medulla magnified about 30 diameters, composed of almost entirely black degenerated fibres, lying between the olive and the pyramid. (4) Fillet above the lesion on the opposite side, and stained very black on account of the large number of degenerated fibres. Section from the upper part of pons varolii.

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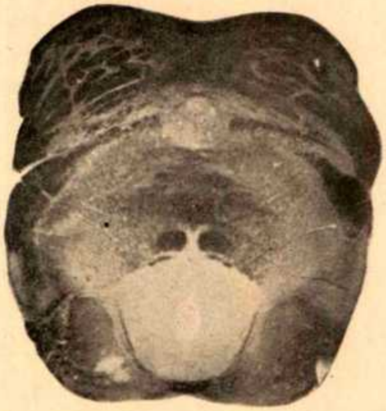
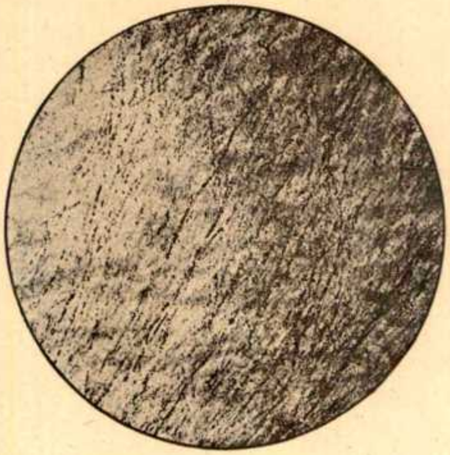
PLATE II. A.



5.



6.

Degeneration of fillet following lesions of posterior column nuclei. (5) Section of the pons at junction with crus cerebri magnified five diameters. The degeneration of the fillet in the section is quite visible to the naked eye, but it is extremely difficult to photograph with such low magnification, but still it will be observed that the fillet is much blacker on one side than the other. (6) Section of optic thalamus showing degenerated fibres of fillet now taking a longitudinal and spreading course to end in the grey matter of this ganglion.

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of the auditory nerve, and these fibres which degenerate downwards in the cord convey impulses from that nucleus connected with equilibration down to the anterior horn cells of the lumbo-sacral region, which of course in the monkey, like man, would preside over the muscles connected with movements and position in space. Certainly these fibres do not come *directly* from the cerebellum as Marchi[22] supposed. This degeneration was extremely well marked in one case in which the lesion was extensive and involved Deiter's nucleus and other cranial nuclei, including the sixth (to the injury of which I attributed an upward degeneration in the posterior longitudinal bundle, of the opposite side mainly), extending as high as the third nucleus. *These fibres may also possibly be ground fibres from motor cranial nuclei, particularly the ocular nuclei, and serving as bridges between spinal and cranial nuclei,* in co-ordinate reflex movements directed by vision.

Summary of results are shown in the three figs. 1, 2, 3. but I shall briefly recapitulate the results of the fourteen experiments.

(1) The column of Goll is formed especially by the fifth, sixth and seventh sub-thoracic roots. Section of the fourth sub-thoracic posterior root produced comparatively little degeneration of Goll's column (Experiment VII.), and further experiments which Dr. Sherrington and I have made show, *as I anticipated, that few fibres enter into the formation of Goll's column above the fourth sub-thoracic.* After uncomplicated unilateral section even of a large number of roots, there is no degeneration in the opposite posterior column. The tract of Goll ends in the nucleus of the funiculus gracilis. When this nucleus is destroyed, or the fibres issuing from it separated from the cells of the nucleus, a degeneration occurs in the internal arciform fibres, the opposite inter-olivary layer and fillet, which can be traced *up to the optic thalamus.* It is impossible to injure Goll's nucleus without Burdach's nucleus being involved, and I found experimentally that the greater number of the fibres of the fillet come from the latter nucleus. *In no case could degenerated fibres be traced to the cortex.*

(2) After unilateral section of roots in most instances degenerated fibres were found in the opposite antero-lateral column, but inasmuch as they were not always present in every case, I assume that these fibres come from cells of the grey matter of the same side as the lesion and cross over in the anterior commissure, and it is possible that *cells from which, in all probability, these fibres arise are damaged by vascular changes in the grey matter*—the result of the operation. These fibres form a tract internal to the ventral cerebellar tract, and in no case were they above forty in number even after section of eight posterior sub-thoracic roots. Some few degenerated fibres were observed in most cases in the lateral column of the same side; none, however, in the direct cerebellar tracts *after section of roots below the second lumbar.*

In connection with this subject *vide* a preliminary communication by J. N. Langley[23] and H. K. Anderson.

(3) After median section in the lumbar region for three-quarters of an inch, there was a symmetrical degeneration in the antero-lateral tract of the two sides. The fibres consist of two sets: (1) The ventral cerebellar, much the more numerous and situated most peripherally, which can be traced to the vermis, looping over the fifth nerve to reach the superior cerebellar peduncle, and then descending on its posterior aspect to the middle lobe of the cerebellum. (2) The crossed afferent tract of Gowers and Edinger (function unknown), which, in all probability, arises from cells of the grey matter, its fibres decussating in the anterior commissure. These fibres occupy exactly the same position, but are much more numerous than the small tract which was observed after unilateral section of roots. They can be traced right up the cord and the medulla in the pons varolii, above this lying to the outside of the lateral fillet, and terminating in the corpora quadrigemina, some few apparently extending to the optic thalamus. Ramon y Cajal[24] has shown that fibres arise from cells at the base of the anterior horn, and decussate in the anterior commissure. It is, therefore, possible that these cells are the source of origin of these two ascending tracts.

In one case of median section the last dorsal segment was injured, and on the same side there was a very extensive degeneration of the direct cerebellar tract.

The fact that the *ventral cerebellar tract is equal and symmetrical in these vertical lesions whether the incision occupies the mid-line or not*, shows that it, like the crossed afferent tract of Edinger, *is a crossed cerebellar tract* coming from the cells of the grey matter, possibly at the base of the anterior horn.

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A CASE OF AMYOTROPHIC LATERAL SCLEROSIS WITH DEGENERATION OF THE MOTOR PATH FROM THE CORTEX TO THE PERIPHERY.

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THIS disease is considered by the French neurologists to be a distinct entity, but both Leyden and Gowers look upon it as a form of progressive muscular atrophy. It was first described by Charcot, who assumed that the primary lesion is degeneration of the pyramidal tracts, and that the affection of the grey matter is secondary or deuteropathic, even where the atrophy is atonic. This assumption is held to be unwarranted by Gowers[1] and Leyden, and the former states: "It is probable that the pyramidal tracts are degenerated, if not constantly, at any rate in such a very large proportion of the cases of progressive muscular atrophy, that Charcot's distinction is in effect giving a new name to an old disease." In the case which I am about to describe, the degenerative process seemed to have affected simultaneously and progressively the whole motor tract, viz.: cortex cerebri, pyramidal system, anterior horn cells, anterior roots, and the motor fibres, with corresponding spasmodic condition, and progressive atrophic changes in the muscles. I am led to believe this partly by the clinical phenomena, but more particularly by the condition of degeneration and sclerosis found in the motor tract. The following degenerations in

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[1] "Diseases of the Nervous System," vol. i., "Spinal Cord and Nerves."

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the brain have been described by various observers. *Kahler and Pick*[1] were the first to observe degenerations above the pons; they noted sclerosis in the external part of the middle third of the crus cerebri, and an atrophy of the central convolutions, as evidenced by the increased depth and breadth of the pre-central and central sulci. *Kowjewnikoff*,[2] of Moscow, described a case of amyotrophic lateral sclerosis, in which the degeneration of the fibres of the pyramidal system could be followed by means of granular bodies through to the cortex. In the peduncle, the sclerosis occupied the middle third of the crusta symmetrically on the two sides. Above, in the posterior part of the internal capsule, it was also found, and thence it was followed into the convolutions which border on the fissure of Rolando, being more marked in the ascending frontal than in the ascending parietal. He also noted a naked eye shrinking of the ascending frontal and of the lower part of the ascending parietal convolutions. *Charcot and P. Marie*[3] have confirmed the existence of these cortical lesions; they have noted also the disappearance of the large motor cells of the cortex, at the level of the para-central lobule and the intracerebral lesion of the centrum ovale, corresponding to the pyramidal bundle. In all these situations they have found granular bodies. *Lombroso*[4] has confirmed the existence of these granular bodies along the intracerebral tract of the pyramidal bundle. *Joffroy et Achard*[5] have described a case in which the central lesions were limited to the spinal cord and bulb with peripheral neuritis and muscular atrophy in the lower limbs.

Rovighi et Melotti[6] noted the integrity of the internal capsule and were only able to find degeneration starting from the peduncles.

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[1] "Beitrage zur Pathologie und Pathologischen Anatomie des Central Nervens Systems," 1879, p. 157.

[2] *Centralblatt für Nervenheilkunde*, 1885.

[3] "Deux nouveaux cas de sclèrose latérale amyotrophique," *Archive de Neurologie*, 1885, tome x., p. 1.

[4] *Lo Sperimentale*, 1888.

[5] *Archive de Médécine experimentale d'Anatomie et Pathologie*, vol. xi., p. 121, 1890.

[6] "Contributione allo Studio della Sclerose laterale Amiotrofica," *Revista sperimentale di Frenatria*, 1888, tome xiv., p. 315.

Paul Marie[1] has described a case of amyotrophic lateral sclerosis without any lesion of the pyramidal bundle at the level of the pyramids. Tooth[2] and Turner could not give any positive opinion, owing to decomposition of the internal capsule and crus cerebri, and this paper deals more with the origin of the cranial nerves.

In the spinal cord the sclerosis has been found limited to the tracts which are motor in function, viz., the crossed and direct pyramidal. The latter varies in size in different individuals, and therefore the sclerosis varies proportionally. Various observers have described a degeneration of the anterior ground bundle. Mœli, Leyden, Flechsig, Charcot and others, and Muratoff,[3] who quotes these authorities found that in all the cases where the anterior ground bundle is sclerosed, the posterior longitudinal bundle was also affected. This favours Flechsig's view that these two tracts are functionally analogous. A slight degeneration of the fillet has been observed in one case by Kronthal,[4] and in another by Muratoff.[5]

An important case has lately been described by Senator.[6] A woman who had all the symptoms of amyotrophic lateral sclerosis died. At the autopsy atrophy of anterior horn cells observed, but no sclerosis of lateral columns. There was renal atrophy and chronic vascular degeneration.

Tooth and Turner (*loc. cit.*) say: "It must be borne in mind that in many instances no change has been found in the peripheral nervous system." I am, however, of opinion that this statement is liable to fallacious interpretation. If the anterior horn cells are destroyed, the axis cylinder processes which form anterior roots and motor efferent fibres must perish; but it is possible that they leave no trace of their previous existence behind. With regard to the anterior horn cells, it is especially the anterior and internal groups that are destroyed, the posterior and external to a less degree, and Clarke's column is usually not affected.

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[1] *Archive de Neurologie*, 1887, tome xiii., p. 387

[2] "Study of a Case of Bulbar Paralysis with Notes of the origin of certain Cranial Nerves," BRAIN, part. lvi., 1891.

[3] Muratoff, *Neurol. Centralblatt*, 1891.

[4] Kronthal, *Neurolog. Centralblatt*, 1891.

[5] Muratoff, *Neurolog. Centralblatt*, 1891.

[6] *Wiener, Med. Wochenschrift*, Nov. 31, 1894.

I will now give the clinical history, notes of autopsy and microscopical examination of my case, and I take the opportunity of expressing my indebtedness to Dr. Bruce for kindly allowing me to see the case in consultation with him, and also allowing me the use of the notes for publication.

Summary of Clinical Phenomena.—One year's duration. Commenced with weakness and numbness in right leg, gradually increasing, and attended with wasting of muscles and exaggerated deep reflexes followed by similar affection of right arm and hand, with especial wasting of thenar, hypothenar eminences and interossei. A little later left leg affected in a similar manner to the right, and then left arm and trunk. Terminated with bulbar symptoms, and probably paresis of diaphragm.

Sarah Albrow, age 39, dressmaker, admitted into Charing Cross Hospital, January 18, 1894. She had been seen on May 1, 1893, by Dr. Bruce, who made the following notes:—

Nervous debility in family. No previous illness of a serious nature, but for twelve months past her health had been failing. For the last five months she had been troubled with a feeling of cold in the right leg, from knee to toes, and a sense of weight in walking. The difficulty increased so that she dragged the right leg, which felt heavy and cold, and it commenced to waste. The right arm began to fail twenty-one days ago; gradually progressive weakness came on, just like the leg, but especially in the thumb and forefingers, and she experienced a difficulty in holding things. Exaggerated knee-jerks present on both sides, and ankle clonus especially marked on the right side. Sensation in no way affected. All the muscles of the upper limbs most irritable on tapping. Vision quite good. Hearing good. Visibly lame in right foot, shuffles it along, especially front part of it. Nothing definitely abnormal, pulmonary, or cardiac. Urine said to be normal. The patient was again seen by Dr. Bruce, and recommended to come into the hospital. The following notes were taken while she was in the hospital:—

Heredity.—Father and mother alive and healthy; brothers and sisters ditto.

Past Personal History.—Scarlet fever and measles when a child. From that time up to the commencement of present illness patient was quite well. Up to the time of the present illness patient menstruated regularly.

Present Illness.—Commenced about ten months ago. Previous to this for about three months patient suffered with severe occipital neuralgia, and a feeling of languor and tiredness. This was followed by numbness, coldness and weakness in the *right leg*, so that she dragged it; then the *right arm* became affected in a similar manner. Later the *left leg* became affected, and finally the *left arm*.

For the last month she has been unable to stand or walk, owing to complete loss of power in the legs, and there has supervened a progressive weakness of the muscles of the back and neck, so that she is unable to hold her head up.

Present Condition. — Consciousness, comprehension, reason and memory good; no fits.

Spine.—Some tenderness in the cervical region.

Motion.—There is complete loss of power in the legs and arms. There is great wasting of the muscles of the upper and lower limbs and trunks, especially the small muscles of the hands, and the extensors of the forearm, the biceps and the deltoid, the small muscles of the feet, and the flexors of the knee and ankle. The intercostals and pectorals are also wasted.

There is a want of mobility of expression in the face, and this is more marked on the right side. Tongue is protruded in the mid line, and there is no fibrillary contraction. Her speech is somewhat slow, but there is no other impairment, and she is able to swallow perfectly well. Fibrillary contraction may be observed in the limbs and trunk. She lies on her back, with the arms adducted to the sides of the body. The forearm is semi-flexed in pronation; the wrist is flexed. There is marked wasting of the thenar and hypothenar eminences, and of the interossei, and the atrophy bears the Aran-Duchenne type. There is considerable rigidity in the shoulder, elbow and wrist, and movement causes pain. *Deep reflexes.* Elbow tap exaggerated on left side, and also on right, but much less marked.

Knee-jerk exaggerated on both sides.

There is a good deal of rigidity in the neck, and pain on movement.

The sphincters are unaffected, and all the special senses are perfect. There is no loss of sensibility to pain nor heat and cold.

Urine, no albumin, no sugar.

The wasting progressed daily until she became a living skeleton unable to move, and the breathing was mostly abdominal.

ELECTRICAL EXAMINATION.

A. Faradic Current.

Electric sensibility appears to be markedly increased.

Left leg.

The muscles of the leg do not react to a strong current.

To the very strongest current the

Extensor brevis digitorum gives a sluggish and feeble contraction.

The *peroneus longus* and *brevis* give only a faint flicker to the very strongest current.

There is no reaction in the muscles of the calf in either leg to the strongest current.

Right leg.

None of the muscles react to a strong current.

To the strongest current the

Flexor longus digitorum contracts sluggishly but fairly forcibly, and

The *extensor brevis digitorum* gives a faint and sluggish contraction.

The *dorsal interossei* on both feet do not contract to the strongest current.

Left thigh.

The extensor muscles, and notably the *vasti* contract feebly to a moderate current.

The *sartorius* gives a sluggish and feeble contraction.

There is no evidence of contraction in the hamstrings or adductor muscles, even to the strongest current.

Right thigh.

The *vastus internus and externus* react fairly to a moderate current, and slightly more than on the left side.

The flexor muscles and adductors do not contract.

The application of the pole upon the extensor muscles of both thighs causes marked patellar clonus.

Left arm.

The *brachialis anticus* and *biceps* respond to a strong current.

To the same current the *triceps* only gives a faint flicker, as also the *palmaris longus*.

Forearm.

Flexor profundus digitorum contracts sluggishly and feebly

Flexor sublimis digitorum contracts sluggishly and feebly

Flexor longus pollicis contracts well by comparison.

There is no contraction of any of the extensor muscles of the forearm.

Hand.

The dorsal interossei do not react to the strongest current.

The *abductor minimi digiti* gives a faint flicker, and the *flexor brevis pollicis* (the internal head) gives a forcible but very sluggish contraction.

Right arm.

To strongest current

Biceps and brachialis anticus, reaction nil.

Triceps, no reaction.

Forearm.

Flexor profundus and sublimis give faint and sluggish contraction, with a strong current, as also the *flexor carpi ulnaris*.

No reaction in the flexor longus pollicis.

Hand.

The flexor brevis pollicis contracts forcibly but sluggishly.

None of the other muscles of the hand react even to the very strongest current.

B. GALVANIC CURRENT.

Left leg and thigh.

Gastrocnemius and soleus contraction very weak. ACC > KCC.

Tibialis anticus. ACC > KCC.

Peronei. ACC > KCC.

No response whatever in extensor communis digitorum.

Extensor brevis digitorum normal.

Extensor muscles of thighs normal.

No response whatever in the hamstrings.

Right leg and thigh.

No contractions whatever in *gastroc. and soleus.*

Flexor longus hallucis. ACC > KCC.

Tibialis anticus. ACC > KCC.

Extensor brevis digitorum, no reaction, remaining muscles of leg normal, but very weak contraction.

Vastus internus, externus, and the rectus, contract normally and fairly strongly.

No reaction whatever in the hamstring muscles.

Left arm.

Biceps and brachialis anticus normal, but weak contraction.

Triceps no reaction.

Forearm.

Flexor profundus digitorum. ACC > KCC.

Flexor carpi ulnaris, normal, but very weak; no reaction in the extensor muscles.

No response in muscles of hand.

Right arm.

Biceps and brachialis anticus, normal, but very weak, no reaction whatever in triceps.

Forearm.

Flexor carpi ulnaris. ACC > KCC.

Flexor profundus digitorum. ACC. > KCC.

No reaction in the extensor muscles.

No reaction in any of the muscles of the hand.

SUMMARY OF ELECTRICAL EXAMINATION.

Extensor muscles of the thigh preserve for the most part their electric excitability.

The *extensor muscles* of the leg are electrically stronger than the flexors.

In the arms the flexor muscles are the stronger; the only muscles in the forearm which react at all well are the *flexor carpi ulnaris*, and the *flexor profundus digitorum.*

The muscles of the hands and feet appear to have lost almost entirely electric sensibility to both currents.

It was thought by the house physician, after she had been in the hospital a fortnight or three weeks, that there was some improvement in the movement of the legs; doubtless it was due to some of the spasmodic rigidity having become less.

She was troubled with pains in the limbs, waking her up from her sleep, otherwise she seemed no worse in her general health.

March 19.—At 10 p.m., patient had a new development of symptoms beginning with a sudden fit of coughing, followed by shortness of breath, and inability to sleep owing to the repeated attacks of coughing. When she was examined it was found that there was considerable dyspnœa and cyanosis of lips, the respirations 48, short and jerky. The diaphragm was overacting, and there was hardly any intercostal movement. In the lower part of the chest the interspaces were retracted with each inspiration. The right sterno-mastoid was not acting so well as the left. Pulse 108. Temperature normal.

Very little air is entering the left lung. Patient complains of a dull aching pain between the shoulders and in the legs. Oxygen was administered with an inhaler, but without much relief. Injection of morphia and strychnia ℳ 1½ of each was administered with benefit.

March 20.—The difficulty in breathing is diminished and she is less cyanosed. The left side of the chest hardly expands at all, the lower intercostal spaces are retracted, and the diaphragm is doing all the work.

March 21.—Respiration is quieter and less jerky, and appears to be carried on solely by the diaphragm. Patient's speech is rather indistinct, and she does not swallow so well.

March 22.—Respiration 30. Pulse 90. Her speech less distinct.

April 3.—Last night patient was suddenly seized with cough and a sense of oppression in breathing. The alæ nasi are working. Respiration 44. Marked cyanosis. Speech greatly affected, unable to swallow solid food, and nervous about taking liquids. Saliva dribbles from the angle of the mouth. No air entering left lung. The left side of chest does not move. Temperature 101·4°. Strychnia and morphia injections repeated. Her condition became worse and worse, and she died on April 4.

Autopsy.—Twenty-one hours after death by Dr. Arkle, who made the following notes :—

The body is greatly emaciated, rigor mortis well marked. Weather temperate. The body lies on the heels, buttocks and shoulders, owing to the arching of the back. Slight *post-mortem* staining of dependent parts of body. The hands are smooth and glossy, and the fingers are over extended and cannot be flexed. There is a marked wasting of all the small muscles of the hand and forearm, the thenar and hypothenar eminences. Toes of both feet flexed and ankle joints of both feet extended. Skin of feet smooth and shining. There is a very small abrasion the size of a lentil over the sacral region. On opening the chest, the following points were noticed: The lungs are very soft, congested, and there are caseous foci at both apices. Heart 7 oz., very small, otherwise normal to the naked eye. Abdomen—liver 40½ oz. normal, spleen 2½ oz., very small and pale. Kidneys weigh together, 7½ oz., small, red in colour, capsule very adherent.

Central Nervous System.—On removal of calvarium a good deal of blood escaped, the dura mater appears somewhat thickened. The longitudinal sinus contains a little fluid blood. There is an opacity and thickening of the arachnoid and pia mater, especially about the central convolutions. The Pacchionian bodies are unusually large. There is no thrombosis or blocking of the large vessels, nor any evidence of softening or growth. On slicing the cerebrum a great deal of congestion and increased vascularity was observed both in the grey and white substance, but no other naked eye hæmorrhage.

Spinal Cord.—On opening the spinal canal, an opacity and yellowish appearance of the arachnoid and pia mater presents itself, especially in the cervical and lumbo-sacral enlargements.

The Cord.—On cutting the cord it was noticed to be softer than natural in the dorsal region. The whole of the central nervous system and the following nerves, phrenics, vagus ulnar, median and sciatic nerves were placed in Müller's fluid for hardening.

After hardening the brain in Müller's fluid and transferring to spirit, I noticed that the ascending frontal and ascending parietal convolutions were shrunken below the level of the other convolutions, and the central and precentral sulci were deeper and broader than usual.

Microscopical Examination of the Central Nervous System.—After hardening in Müller's fluid, my methods of examination were as follows:—Pieces of tissue were stained by the Marchi method and embedded in celloidin. Other portions were placed in spirit and then embedded in celloidin, and sections of the same were cut and stained by Weigert-Pal carmine method and with hæmatoxylin and eosin.

Condition of Membranes and Vessels.—The pia mater is considerably thickened, in places laminated; the vessels are gorged with blood, and in their neighbourhood are enormous numbers of leucocytes. The neuroglia tissue is increased, and this is especially evident in sections of the cortex of the central convolutions just beneath the surface, the network of connective tissue being continuous with and extending from the pia mater. Sections of the brain and spinal cord stained with hæmatoxylin and eosin with a view to microscopical examination of the condition of the membranes and vessels exhibited the following points:—Thickening of the pia mater and marked congestion of the capillaries, small arteries and veins. The membranes covering the surface of the brain and dipping into the sulci exhibit a condition of chronic peri-arterial inflammation, being infiltrated with leucocytes, and in places laminated. The walls of the small vessels are thickened, and the muscular coat of the arterioles is somewhat hyaline in appearance. There are numerous small hæmorrhages. This condition of the membranes and vessels appears to be more manifest over the central convolutions than in the occipital lobes. The membranes and vessels of the spinal cord present a similar appearance, but not so marked. I regret that the kidneys were not preserved for examination. Their naked eye appearance and size suggested a chronic vascular degenerative change with overgrowth of connective tissue.

Brain and Internal Capsule.—Sections of the cortex of the *central convolutions* showed numbers of granulation corpuscles, whereas sections of the occipital lobes showed none.

Sections of the cortex of the central convolutions stained by Marchi's method showed that numbers of nerve fibres had disappeared, also that numbers of fibres were undergoing degeneration. This was also manifest by the Weigert or Pal method, but the degenerating fibres cannot be seen nearly so definitely. *Vide* photo-micro., Plate III. (1). Sections of the occipital lobe showed no such degenerated fibres. Examination of a large number of sections revealed absence of the large pyramidal cells. The internal capsule showed a number of degenerated fibres stained black by Marchi fluid amidst empty spaces and sclerotic tissue. These degenerative appearances are seen in the posterior part of the internal capsule. *Vide* photo-micro., Plate III. (2), low power; the black dots are the degenerated fibres in cross section.

Crus Cerebri. — Sections were stained by all three methods with the same result, viz., sclerosis of the whole middle third, and amidst the sclerosed tissue black degenerated fibres can be seen by Marchi staining. *Vide* photo-micro., Plate III. (3), of a section of the crus cerebri stained by Weigert method. The middle portion is quite light, owing to its not being stained by the hæmatoxylin.

Pons.—The pyramidal fibres exhibit extreme degeneration and sclerosis. Fillet and superior cerebellar peduncles unchanged.

Medulla. — Pyramidal fibres, extreme sclerosis and degeneration. *Vide* photo-micro., Plate III. (4). Internal and external arciform fibres and interolivary layer normal. Slight sclerosis of posterior longitudinal bundle. Restiform body normal. Ventral cerebellar fibres degenerated (?). As these sections were only stained by Weigert method, I cannot say for certain whether this is so or not, but certainly the ventral cerebellar tract amidst the external arciform fibres does seem to have more neuroglia tissue than normal.

Nuclei of Grey Matter.—The only nuclei that seem to be degenerated are the hypoglossal and the lower facial with which it is associated, and also the spinal accessory. Many of the cells have disappeared, and some are shrunken and pigmented.

Medulla, at the lower part of the decussation of the pyramids, stained by Marchi's method, showed a sclerosis, with numbers of black degenerated fibres just similar to the appearance presented in the cortex and internal capsule.

Cervical Spinal Cord showed atrophy of anterior horns, with disappearance of the anterior and internal groups of nerve cells, and to a less degree atrophy of the posterior and external groups. Sclerosis of the direct and crossed pyramidal tracts, and considerable atrophy of the ground fibres. The direct cerebellar tract, lying outside the crossed pyramidal tracts, is normal. *Vide* photo-micro. (5), Plate IV.

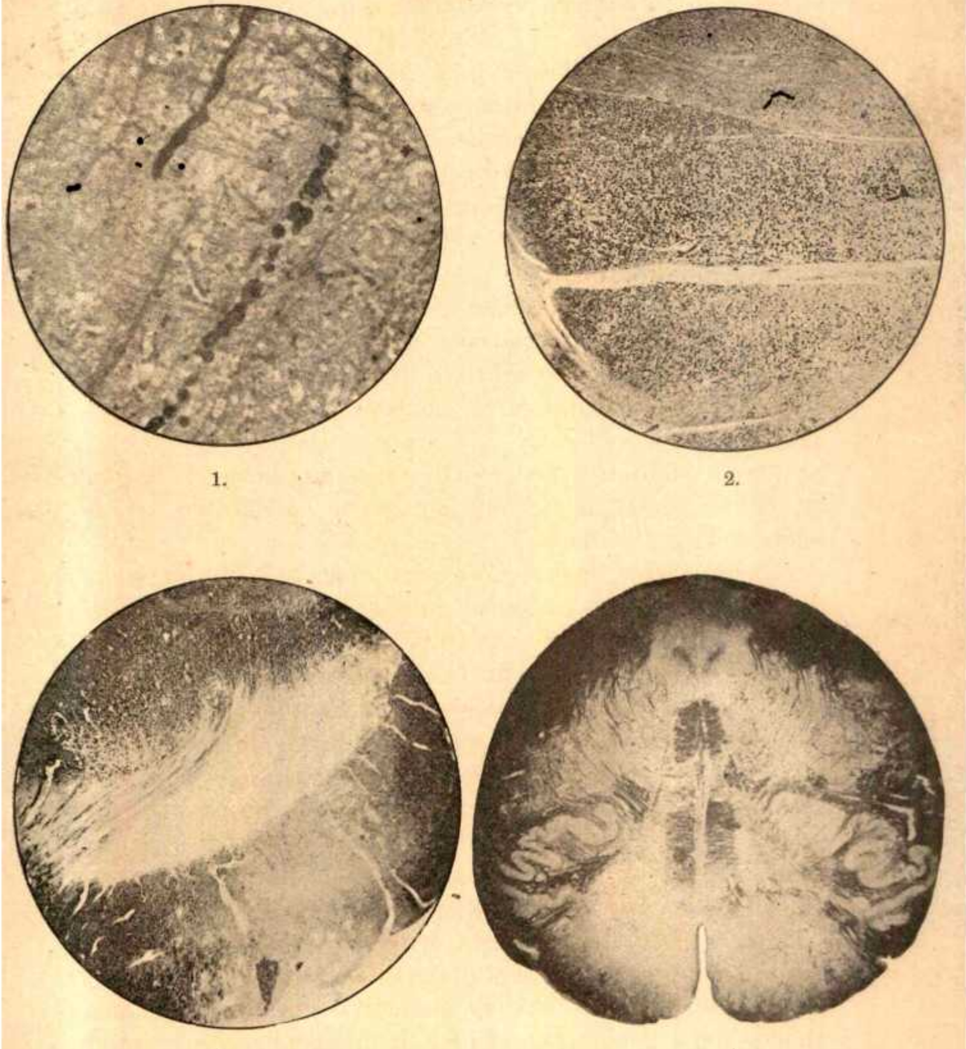
Dorsal Spinal Cord.—The anterior horn cells are atrophied. Clarke's column apparently unaffected. *Vide* photo-micro. (6), Plate IV.

Lumbo-sacral Spinal Cord and Roots.—Posterior roots normal. Anterior roots, great outfall of fibres. *Vide* photo-micro (10). Here again the anterior and internal groups of cells are most atrophied but the posterior and external group, although they have not disappeared, are undoubtedly undergoing degeneration. *Vide* photo-micro. (7), Plate IV., of this group of cells stained by Marchi's method. The cells are black, presumably from fatty degeneration (this is usually termed pigment, but it stains black with osmic acid). Many of them have their processes gone; some are shrunken, and some have swollen up.

Nerves.—Phrenics stained with osmic acid and picro-carmine showed numbers of fibres undergoing degeneration. Vagus, treated in a similar manner, showed some fibres undergoing degeneration. Ulnar, median and sciatic showed very few fibres undergoing degeneration.

Sections of these nerves, however, stained by osmic acid and cut in celloidin, showed THAT A LARGE NUMBER OF FIBRES HAD BEEN DESTROYED, and many of the bundles were much smaller, so that a section of the ulnar, when

PLATE III.



Amyotrophic lateral sclerosis. (1) Degenerated fibres in cortex cerebri. (2) Transverse section of degenerated fibres in the anterior part of the posterior half of the internal capsule, both stained by Marchi's method. (3) Crus cerebri stained by Weigert's method, showing sclerosis of the middle third. (4) Medulla showing sclerosis of the pyramids also stained by Marchi's method. Photo-micrographs of untouched negatives.

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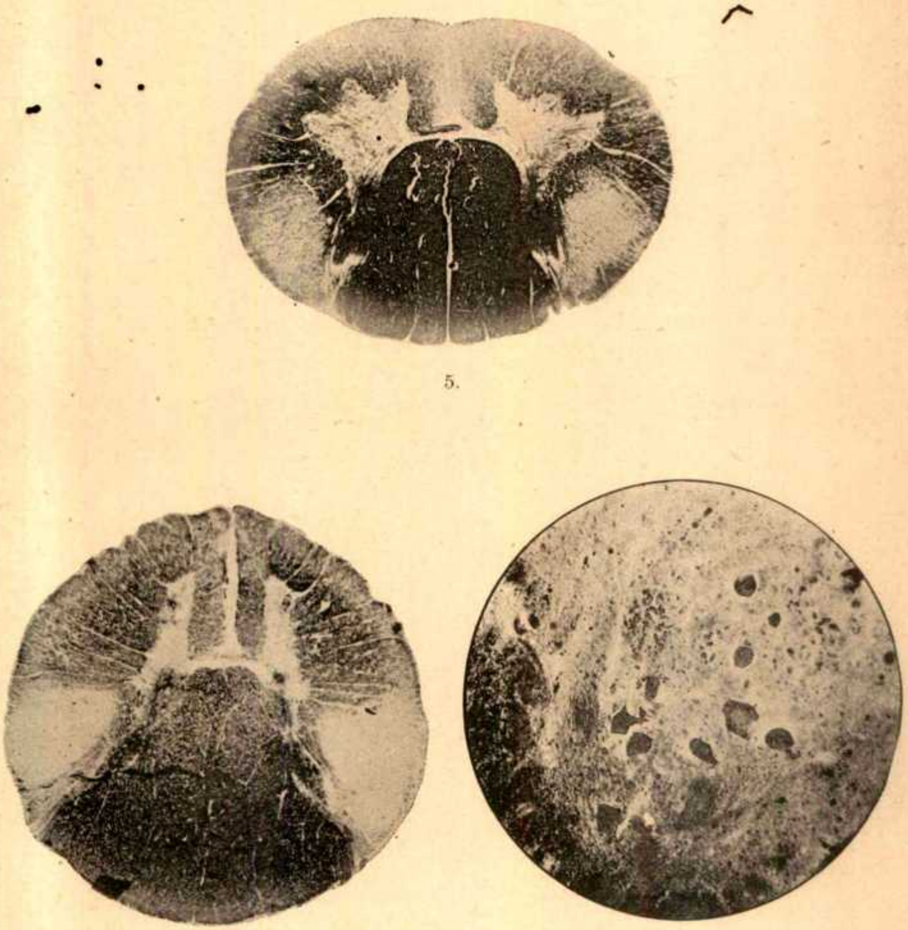
PLATE IV.



5.

6.

7.

Amyotrophic lateral sclerosis. (5) Spinal cord lower part of cervical enlargement. Sclerosis of pyramidal tracts and antero-lateral region. Direct cerebellar fibres seen unaffected outside crossed pyramidal tracts. (6) Spinal cord eighth, dorsal. (7) Posterior external group of cells of anterior horn in lower lumbar region, showing degenerative changes; the anterior internal groups have entirely disappeared in this section.

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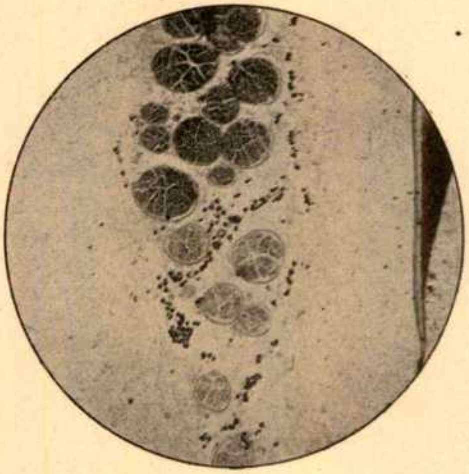
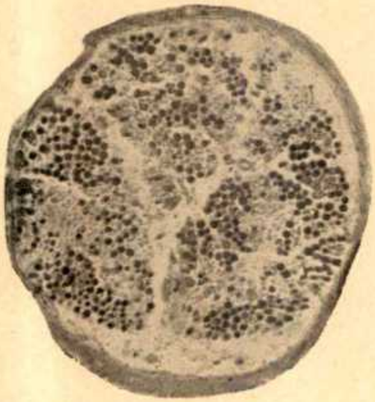
PLATE V.



8.



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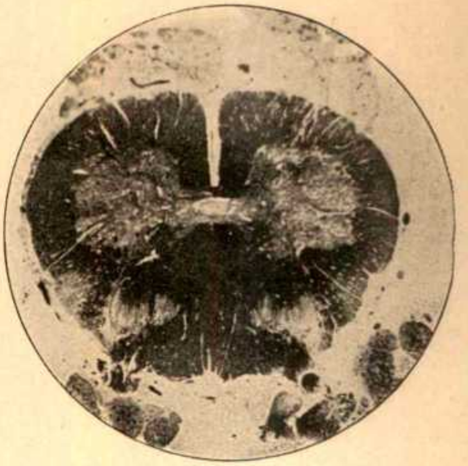

10.

(8) Ulnar nerve from the middle of the forearm, magnified about ten diameters, stained with osmic acid and cut in paraffin. The bundles in the upper portion are normal. The lower six bundles are greatly wasted and do not stain black. (9) One of these bundles highly magnified, shewing that a large number of fibres have completely atrophied. (10) Section of spinal cord in sacral region showing sclerosed crossed pyramidal tracts. Also compare colour of anterior and posterior roots, the former being degenerated.

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looked at with the naked eye, appeared much blacker and fuller in one half of the section than the other. *Vide* photo-micro., Plate V. (8) and (9). In fact, the section appeared to be divided into two parts, one in which the bundles of nerve fibres had shrunk to half their size, and another which was hardly affected at all. The photo-micro. (9) shows one of the sclerosed bundles of fibres. The sclerosis was less apparent in the median and sciatic nerves than in the ulnar. The appearance closely resembles the photo-micrograph of Dr. Sherrington's after section of ventral and dorsal roots, the latter cut proximal to the ganglion. Dr. Sherrington has, in a most valuable paper in the *Journal of Physiology*, October, 1894, shown that a nerve entering a muscle contains a large number of *afferent* muscular fibres. It would be interesting in any future case of amyotrophic sclerosis to examine a so-called purely motor nerve entering muscle to see if these afferent motor fibres are present. It may be that the half of the nerve undegenerated is purely sensory, while the other half so obviously atrophied is muscular, the undegenerated fibres being those afferent muscular fibres of Sherrington. This is very probable, as in the monkey I have found that the ulnar nerve can be split into two portions, one which by stimulation, either by faradism or ligature, is followed by muscular contraction, and another, presumably cutaneous, which gives no such result.

This case has many points of interest. I believe that a simultaneous degeneration of the upper and lower segments of the motor path took place, and the clinical history supports this opinion, for there was wasting of muscles appearing at the same time as exaggerated reflexes. The hemiplegic onset of first the right leg, then the right arm, and subsequently left leg, then left arm, supports the view held by Charcot that the disease is primarily in the upper segment; but against this we have the fact that there was muscular wasting of the typical progressive muscular atrophy type associated with the spasmodic conditions. Are we to believe that the degeneration of the upper segment of the motor path begins in the terminations of the crossed pyramidal tracts in the spinal cord and spreads gradually upwards? Or is it due to degenerative changes in the cortex[1] leading to degeneration and atrophy of the large cells of the third layer which give origin by their axis cylinder processes to the fibres of the pyramidal tract? There is nothing against the latter view in this case, at any rate, although cases in which no degeneration can be found above the pons would support the former view. My own experience of experimental degenerations of the cross pyramidal tracts in monkeys produced by various injuries of the cortex, ablation, subcortical section, and cauterization, lead me to believe that if the connection of the neuron with its cell be destroyed, a degeneration occurs throughout the whole pyramidal system, and does not creep down nor creep up the fibres forming it. It may, however, be different in the case of a slow degenerative process affecting the protoplasmic processes of the cells, which, as Golgi and Ramon y Cajal have shown, bear a most complex arrangement, *like a tree*, and we can easily understand how a chronic peri-arteritis could interfere with the nutrition of the cell, specially as we are led to believe that these dendritic processes project into corresponding lymph spaces which are in connection with peri-vascular lymphatics. Interference by chronic inflammatory changes, such as are found in this case, might very well cause nutritional changes in the cell, eventually leading to its complete atrophy and disappearance. As a rule, the more remote a part is from its seat of nutrition, the more likely is it to undergo degeneration, and we could thus explain the gradual "creeping up" of the degenerative process.

Certainly the appearances of degeneration and sclerosis presented by the internal capsule and the crus cerebri in this case were quite as advanced as the pyramidal tracts of the medulla and dorsal region of the cord.

Of course in the white matter of the hemispheres of the centrum ovale, the process of degeneration could only be seen by scattered degenerated motor fibres and granulation corpuscles, and owing to the large number of intermingled healthy fibres not belonging to the pyramidal system, it is difficult to recognise sclerosis, although when portions of the cortex of the motor area are stained by the Weigert method and compared with the occipital region, there is a marked difference in the appearance of the fibres of the sections of these two areas. By Marchi's method, however, degenerated fibres, such as shown in the photo-micrograph, could be found throughout the sections of the motor area (especially numerous in parts where the disease was not so advanced as in the lower part of the Rolandic area), but none in sections of the occipital, a purely sensory area. Where the pyramidal tract occupies a small area, as from the internal capsule downwards, the degeneration after staining the sections, is apparent to the naked eye.

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[1] This view is supported by the cases of Kahler and Pick, Kowjewnikoff Lombroso, and Charcot and Marie.

It is difficult to understand why, in the spinal cord, the anterior horns alone should suffer, and why particular groups in particular regions—such as the cervical and lumbar enlargements, and the *anterior and internal groups of cells*—be completely destroyed, *while the posterior and external escape;*[1] why the posterior vesicular column, and the direct cerebellar tract which arises from it should be unaffected, if this disease depended solely upon a chronic inflammatory process. If, however, we look upon this progressive muscular atrophy as a degenerative process of the motor path, affecting in some cases the lower segment first, therefore masking the symptoms which would be produced by the subsequent affection of the upper segment, or, in some cases, in the upper segment first, producing characteristic symptoms, or affecting as in this case both segments of the motor path simultaneously; we must then conclude that there is

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[1] Is it possible that the escape of the extensor muscles of the thigh and leg may be associated with the preservation of the posterior and external groups of large multipolar cells in the lumbo-sacral region? The preservation of these cells and the disappearance of the cells of the opposing ham-string muscles which were found to have lost all electrical excitability (and presumably, therefore, innervated by cells of the anterior internal groups which had disappeared) would account for the exaggeration of the knee-jerk which was noticed, even within a month or two of the patient's death. This fact might also serve to account for the exaggerated tendon reflexes observed in Senator's case where the *post-mortem* examination revealed no sclerosis of the pyramidal tracts

DEFECTIVE DEVELOPMENT OF THE CENTRAL NERVOUS SYSTEM IN A CAT.

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Introductory Remarks.

I AM induced to place this case of defective development of the central nervous system in a cat on record, because it appears to me to be of more than ordinary interest.

My grateful acknowledgments are due to Professor Victor Horsley for enabling me to carry out the examination of this animal at the Pathological Laboratory of University College. I am also indebted to Professor Boyce for his kindness in bringing to my aid his large experience of the topographical anatomy of the central nervous system of the cat.

It is impossible for me to express too cordially my thanks to Dr. S. W. Carruthers, to whose generosity I am indebted for the valuable animal on which these observations have been made. The symptoms presented by the cat, during life, reminded Dr. Carruthers of some dogs which I had shown at a meeting of the Neurological Society, and on whom I had practised ablation of one lateral lobe of the cerebellum; he therefore showed the cat at a meeting of the same Society, and afterwards gave it to me, to make use of as I thought fit.

On examining the animal, I found that there was evidence of paresis of both posterior extremities and of the right anterior extremity, which condition corresponded to that met with in dogs after ablation of the right lateral lobe of the cerebellum. It therefore seemed probable that in this cat there must be some lesion, or defect, of the right lateral lobe of the cerebellum. And as will be seen subsequently, the condition found at the autopsy amply confirmed the surmise.

The cat was placed under the influence of chloroform, and killed by an over dose of this anæsthetic. On *post-mortem* examination the skull presented no abnormal features, and, as far as could be ascertained, there was no asymmetry. The membranes were normal, and not unduly adherent to the skull or to the brain. The right cerebral hemisphere appeared to be generally a little smaller than the left, but there was no special defect in any one part of it. The whole brain was below the average size; but there was no evidence of special atrophy or defective development of any part in particular, with the exception of the right hemisphere being a little smaller than the left, as has already been said.

The feature which attracted most attention was the condition of the cerebellum; the whole organ was smaller than normal, and also proportionally, as compared with the cerebrum; but most striking of all was the fact that the right lobe was scarcely a third the size of the left (see fig. 1) The right lobe was diminished in all its dimensions; and the corresponding right half of the pons and medulla shared in its defect, though not to such a marked extent. There was apparently a complete absence of the transverse fibres of the pons on the right side, and the whole of the right side of the pons and medulla were markedly smaller than the left. As far as could be ascertained, this asymmetry was not shared by the spinal cord, the two lateral halves of which appeared to be quite equal.

The cranial nerves were smaller on the right side than on the left; but the spinal ones did not present any detectable difference.

The whole central nervous system was preserved in Müller's fluid for subsequent microscopical examination; the result of which will now be detailed.

Microscopical Examination.

The stain that was chiefly employed in this investigation was Weigert's hæmatoxylin method as modified by Pal, and as further modified by Schäfer. This was variously supplemented by carmine, nigrosin, Delafield's hæmatoxylin, eosine, &c.

After describing the condition of the cerebrum and cerebellum, it will, I think, be best to describe the other defects in the rest of the central nervous system, as they are met with at certain typical levels of the pons and medulla, and then to give a short summary of all the defects which were found in this animal, before discussing the significance of these defects, and the deductions to be drawn from them.

The Cerebellum.

The defective development of the right lateral lobe of this organ, as compared with its left, was even more strikingly brought out on vertical transverse sections being made. The right half of the middle lobe did not appear to share in this defective development of the lateral lobe. Contrary to what might have been expected, the proportion between the different layers of cells of the cortex of this rudimentary right lobe was not altered, and the normal proportion of grey to the immediately subjacent white matter was preserved. The cells of Purkinje were normal in appearance, and in their arrangement, as were the cells of the granular and molecular layers of this rudimentary portion of the cerebellum. Whereas the corpus dentatum was well marked on the left side, its only representatives, on the right side, were a few irregularly scattered cells in the region where it ought to have been, which region was of course also greatly deficient in the amount of white matter. The total quantity of fibres passing to and from the cortex of the right lateral lobe was of course much below the normal, a fact in keeping with the small amount of cerebellar cortex which existed on this side.

The Cerebrum.

Vertical transverse sections through the hemispheres at different points showed less asymmetry than appeared to exist when the brain was viewed as a whole. The grey and white matter were normally arranged, the normal proportion between the two appeared to be preserved, and there was certainly no evidence of gross structural general or local change in either hemisphere. The cortical cells of the left hemisphere showed no evidence of atrophy, and those of the right side were also free from any such defect.

The basal ganglia appeared to be equally and normally developed on the two sides, and there was only a very slight difference in the size of the internal capsule on the two sides, that on the right appearing to be a little smaller than that on the left. But sections through the crura cerebri presented a very different state of things, for there could be no question that the right crus was considerably smaller than the left (see fig. 2). While this striking difference was seen in the size of the crura, the optic tracts appeared to be normally and equally developed on the two sides, and made the asymmetry in the crura the more evident (see fig. 2).

The Region of the Posterior Corpora Quadrigemina.

The asymmetry at this level is exceedingly marked, many of the structures on the right side being almost completely absent. The most striking differences are to be seen in the fillets, superior cerebellar peduncles, pyramids, and corpora quadrigemina; but in addition to these, certain minor differences on the two sides are evident.

The right corpus quadrigeminum is about a third the size of the left (see fig. 3); and the same proportion exists in the sizes of the right and left fourth nerves, as they are seen decussating in the roof of the aqueduct of Sylvius (see fig. 4). While the superior cerebellar peduncle is well marked on the left side, scarcely any trace of this structure can be made out on the right (see fig. 3). The difference in the size of the descending root of the fifth cranial nerve on the two sides is not so striking, for although that on the right is smaller than that on the left, and its fibres are less regularly arranged (see fig. 3), there is not that pronounced difference which has been noted in connection with other structures already described at this level. Scarcely any trace of fillet fibres exists on the right side, while the left fillet is well developed and stands out prominently (see fig. 3). The pyramid on the right side is extremely diminutive, and contrasts markedly with the well developed left pyramid (see fig. 3).

The Region of Exit of the Sixth and Seventh Cranial Nerves.

Many points call for comment at this level (see fig. 5), and foremost amongst them is the complete absence of the pyramid on the right side, there being no trace of this structure to be seen; while on the left side the pyramid is normal.

The enormous difference in the size of the restiform body on the right side as compared with that on the left is striking, for while the latter is of normal size and appearance, the former is represented by an insignificant bundle of fibres, seen cut transversely and situated just internal to the superficial root of the eighth nerve as it makes its exit. The fibres which represent the right restiform body appear to be normal and are not obviously smaller than those of the left restiform body; but they are more crowded together, there being a marked deficiency in the amount of interstitial tissue. The contrast between the two restiform bodies is better seen at a lower level (as represented in fig. 6), at which level the relative sizes of the ascending root of the fifth nerve and the restiform body, on the two sides, is striking.

To revert to fig. 5, however, in addition to the points already mentioned, we see that the posterior longitudinal bundle is smaller on the right side than on the left. The seventh nerve, seen divided transversely as it lies internally to the sixth nucleus, is distinctly smaller on the right side, its fibres appear smaller and do not stain as well as do those of the left nerve.

The sixth nucleus on the right side is a little smaller than that on the left, though the cells appear to be about the same size in both. Fewer fibres pass from the right sixth nucleus to the opposite posterior longitudinal bundle, than from the left nucleus.

The sixth nerve is slightly smaller on the right side than on the left; but the difference in size is not nearly so striking as is that between the two seventh nerves, the right seventh being notably smaller than the left. A still greater difference in size exists between the eighth nerves; and the accessory nucleus of the eighth is a trifle smaller on the right side.

Practically no difference in the size of the ascending root of the fifth exists on the two sides (see figs. 5 and 6), but the substantia gelatinosa is a little less on the right side as compared with the left. There is a difference in the size of the two superior olives, that on the right side being considerably smaller than that on the left, though its cells do not appear to be small or shrunken.

There is almost a complete absence of any connections between the right side of the pons and the cerebellum.

The Region of the Inferior Olive.

A section through the medulla at this level presents a curious picture (see fig. 7). The pyramid on the left side is well developed and occupies its normal position, whereas on the right side, where the pyramid ought to be, the inferior olive is situated, with the anterior external arcuate fibres, and in addition, what appear to be a few pyramidal fibres forming its inner and anterior boundary. The inferior olive on the left side is exceedingly poorly developed, and represents about a twelfth part of that on the right side in size.

The lateral medullary tract on the right side is almost completely absent, a distinct concavity existing in the periphery of the medulla in this region, instead of the normal convexity which exists on the left side. The nucleus lateralis is also almost completely absent on the right side at this level. No trace of Boyce's direct tract,[1] which degenerates downwards after ablation of the cerebral hemisphere on the same side, is to be seen; while on the left side this tract is so prominent as to suggest the possibility of its being hypertrophied.

The ascending root of the fifth is equally well developed on the two sides; and the substantia gelatinosa is almost equal also, though possibly it is slightly less on the right side. There is not much to choose between the funiculus gracilis on the two sides; but with regard to the funiculus cuneatus, there is a marked difference, that on the left being about half the size of that on the right.

In addition to these differences, there is a general deficiency in the formatio reticularis on the right side.

The Region of the Decussation of the Anterior Pyramids.

The first and most important point to be noted at this level is with regard to the anterior pyramids. Whereas that on the left side, in accordance with what we have seen at higher levels, is well developed, there is only the merest trace of a pyramid on the right side, an insignificant bundle of fibres alone representing this structure at this level. But the point of extreme interest and importance lies in the fact that instead of the whole of the fibres in the well developed left pyramid passing across the middle line to the region of the right crossed pyramidal tract, the pyramid bifurcates, and while the majority of its fibres pass to the region of the opposite crossed pyramidal tract, about a fifth of their number pass to the region of the crossed pyramidal tract of the same side, that is, to the left crossed pyramidal tract (see fig. 8). The few fibres occupying the position of the anterior pyramid on the right side are distributed as follows: some decussate and pass to the opposite crossed pyramidal tract, some pass with the bulk of the fibres from the opposite anterior pyramid to the region of the crossed pyramidal tract of its own side, while some turn forward into the grey matter of the anterior horn (see fig. 9). The next point worthy of note is the fact that there is a distinct deficiency in the region of the direct cerebellar tract on the right side, owing evidently to absence of this tract. Instead of the contour of the cord being preserved, there is a distinct concavity in the periphery at this region, which contrasts obviously with the normal convexity in the same region on the left side.

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[1] Boyce, *Proc. Roy. Soc.*, 1894.

The defective development of the funiculus gracilis and funiculus cuneatus, with their nuclei, on the left side, is better seen at this level than in any that have been examined higher up. The funiculus gracilis is a little more than half the size of that on the right side, while its nucleus is only a third the size of that of the other. It is not so easy to judge of the exact amount of diminution in size of the funiculus cuneatus, but its nucleus is a little more than half the size of the nucleus of the right funiculus cuneatus (see fig. 8).

The Spinal Cord.

It is most surprising that, in spite of the complete absence of the right pyramid at certain levels in the pons and medulla, and the evident defective development of the direct cerebellar tract noted at the level of the decussation of the pyramids, there should be next to no asymmetry of the spinal cord. As may be seen even from fig. 10, which represents the upper cervical part of the cord, it is difficult to say that there is any difference on the two sides, and the same holds good in the dorsal region; but in the lumbar there is possibly some evidence of slight deficiency on the left side, chiefly in the region of the left crossed pyramidal tract. Nowhere, throughout the length of the cord, is there that difference on the two sides, which might have been expected, in view of the defects of those systems at higher levels which we know contribute to the formation of the cord.

No minute microscopic differences can be detected in the nerve fibres on the two sides of the cord, and the grey matter is normal in amount, as compared with the white, and in arrangement. The minute structure of the grey matter is also normal on both sides, the cells being apparently perfectly natural and showing no structural alterations.

The anterior and posterior nerve roots present no differences on the two sides.

Summary.

It will be profitable for us to review briefly the salient abnormal features presented by this animal, before attempting to discuss the possible significance of certain of these.

First we have the fact that the cerebellum is smaller than normal, as a whole; and that its right lateral lobe is very much smaller than its left.

As to the cerebrum we have seen that only slight differences exist on the two sides in which the right is the slightly defective one, that this slight difference is also apparent in the internal capsule, and that when we come to examine the crura cerebri the right is very distinctly smaller than the left.

The pyramids present many features of great interest. The right pyramid is completely absent throughout the medulla, until the lowest part is reached; but at the level of the decussation a few fibres indistinguishable from pyramid fibres are seen occupying the region of the anterior pyramid on the right side. At the decussation, the normal left pyramid bifurcates, and while the greater number of fibres pass across to the opposite crossed pyramidal tract, a considerable number pass to the crossed pyramidal tract of their own side. Compensation is so complete that, with the exception of possibly a slight difference in the lumbar region of the spinal cord, the crossed pyramidal tract does not appear to be smaller on the left side than on the right.

Practically no trace of fillet fibres can be found on the right side.

All the peduncles of the cerebellum are very deficient, there is practically complete absence of the superior and middle cerebellar peduncles on the right side, and the restiform body, or inferior cerebellar peduncle, is exceedingly diminutive on the same side.

The cranial nerves are all smaller on the right side than on the left, though they are not all equally so.

The superior olive is slightly smaller on the right side; but the inferior olive is exceedingly well developed on this side, while that on the left side is extremely poorly developed.

The nucleus of the funiculus gracilis and that of the funiculus cuneatus are smaller on the left side than on the right.

It is thus clear that in a pretty universal defect of development of the right side of the pons and medulla there are certain notable structures which do not share to any extent in this defect; they are the ascending root of the fifth nerve, the substantia gelatinosa, the inferior olive, and the nuclei of the posterior columns. Of these structures, two are not defective on the left side either, viz.: the ascending root of the fifth and the substantia gelatinosa; while the inferior olive is markedly defective on the left side, as are the nuclei of the posterior columns, though to a much smaller extent.

Deductions.

That the diminutive right lateral lobe of the cerebellum is a primary condition the result of defective development, and not one due to atrophy secondary to any other lesion of the central nervous system, is evident from the fact that the portion of cerebellar tissue present is normal in structure, neither the cells nor fibres composing it showing any sign of atrophy or other abnormality. It is as if a small portion of normal cerebellar tissue had been left, and the whole of the remainder of the lobe had been cut away. In this respect this case contrasts markedly with two cases of atrophy of the cerebellum in cats recorded by Herringham and Andrewes.[1] The condition of the cerebellum in their cases was apparently primary also, but instead of the cerebellar tissue present

[1] Herringham & Andrewes, *St. Bartholomew's Hospital Reports*, vol. xxiv.

being of normal structure, there was great atrophy of the granular and molecular layers of the cerebellar cortex. Their cases also differed from mine in that the whole cerebellum participated in the atrophy, and not one lateral lobe more than the rest of the organ.

The defective condition of the cerebellar peduncles on the right side calls for no comment, for that the superior peduncle, the transverse fibres of the pons or middle peduncle, and the restiform body or inferior peduncle should be thus defective is only in keeping with the diminutive size of the right lateral lobe of the cerebellum itself.

With so little cerebellar tissue representing the right lateral lobe, it is quite in accordance with our knowledge of the relationship between one lateral lobe of the cerebellum and the opposite inferior olive that the latter should be so defectively developed. The relationship is one which has been well established by pathological observations, and which has been put beyond question by the results of experiment, for it has been found that after ablation of one lateral lobe of the cerebellum the opposite inferior olive atrophies. In the present case it would appear to be not so much a secondary atrophy of the left inferior olive as a defective development of it in association with the defective development of the right lateral lobe of the cerebellum. The fact that the whole of the left side of the pons and medulla is perfectly normal, with the exception of the inferior olive and posterior column nuclei is a most significant one, and points unquestionably to their defectiveness being related in some way to defect of certain structures on the right side. We shall see presently what the probable significance of the defect of the posterior column nuclei on the left side is, and as to the defect of the inferior olive there can, I think, be no reasonable doubt that it is associated with the defective development of the right lateral lobe of the cerebellum. Similarly the well developed inferior olive on the right side, which is otherwise full of abnormalities, is no doubt consequent on the left lateral lobe of the cerebellum being fairly well developed, when compared to the right; a remark especially applicable to the state of development of the corpus dentatum on the two sides.

This case points to the crossed relationship between one lateral lobe of the cerebellum and the opposite cerebral hemisphere as not being one in which the opposite cerebral hemisphere atrophies, or is defectively developed, in consequence of the abnormality of the lateral lobe of the cerebellum. Congenital cases of atrophy of one lateral lobe of the cerebellum and of the opposite cerebral hemisphere are always open to question as to which is the primary condition and which is the secondary. In thirteen cases, collected by him, Turner[1] concluded that the cerebellar atrophy was secondary to the cerebral. That one lateral lobe of the cerebellum may atrophy in consequence of damage to the opposite cerebral hemisphere is strikingly proved by a case published by Ferrier,[2] in which the one cerebral hemisphere had been converted practically into a cyst, by a hæmorrhage, and in which there was well-marked secondary atrophy of the opposite lateral lobe of the cerebellum. But I know of no similar proof that one cerebral hemisphere ever atrophies in consequence of destruction of the opposite lateral lobe of the cerebellum. A few cases, however, have been recorded in which with well-marked atrophy of one lateral lobe of the cerebellum there has been slight atrophy of the opposite cerebral hemisphere,[3] and Wagner[4] states that he found atrophy of the cerebrum after removal of the cerebellum in pigeons; but this result stands alone, and is open to question. No other observer has recorded atrophy of the cerebrum consequent on ablation of the cerebellum, so far as I am aware; and my own results of extirpation of parts of the cerebellum in dogs forbids my concluding that one hemisphere of the cerebrum atrophies in consequence of destruction of the opposite lateral lobe of the cerebellum. That the one half of the cerebellum exercises a very marked

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[1] Turner, *Schmidt's Jahrb.*, 1856, vol. xc., p. 32.

[2] Ferrier, BRAIN, April, 1882.

[3] *Cf.* Weber, Cramer, &c.

[4] Wagner, *Journ. de Physiol.*, 1861, vol. iv., p. 394.

PLATE TO ILLUSTRATE DEFECTIVE DEVELOPMENT OF THE CENTRAL NERVOUS SYSTEM IN A CAT.

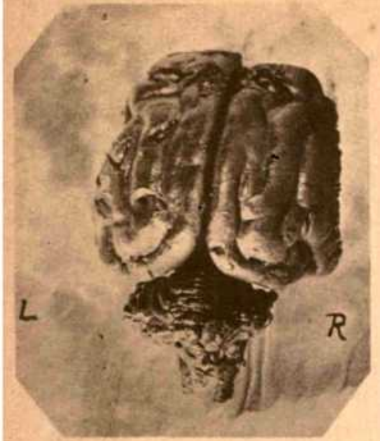

FIG. 1.

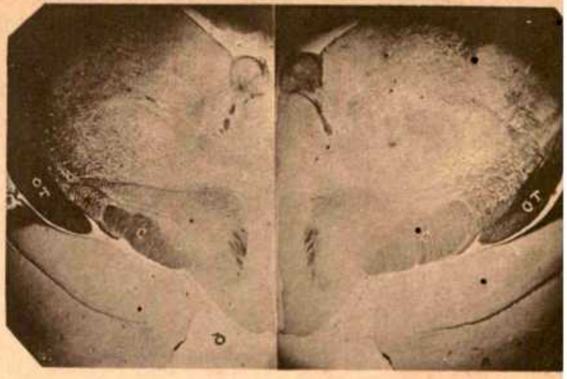

FIG. 2.

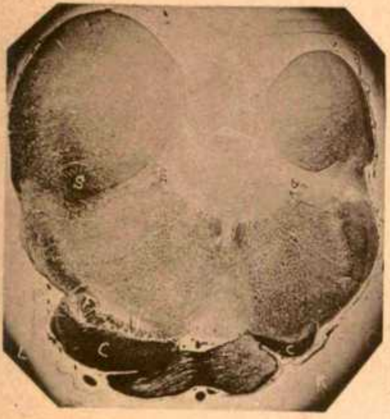

FIG. 3.

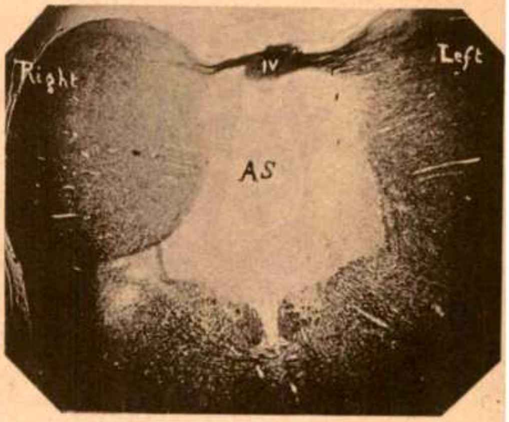

FIG. 4.

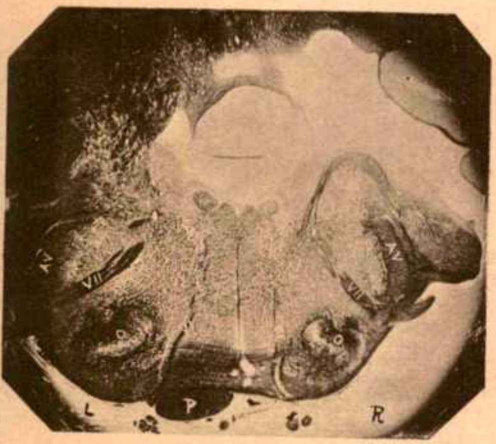

FIG. 5.

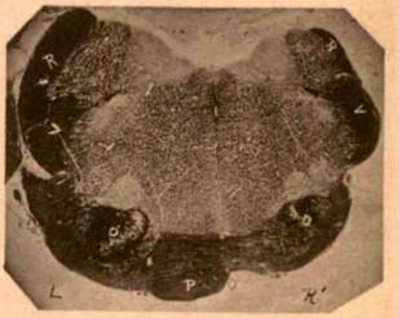

FIG. 6.

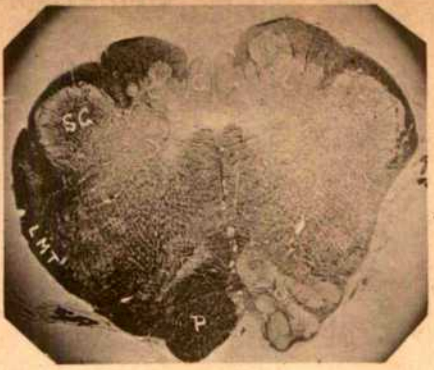

FIG. 7.

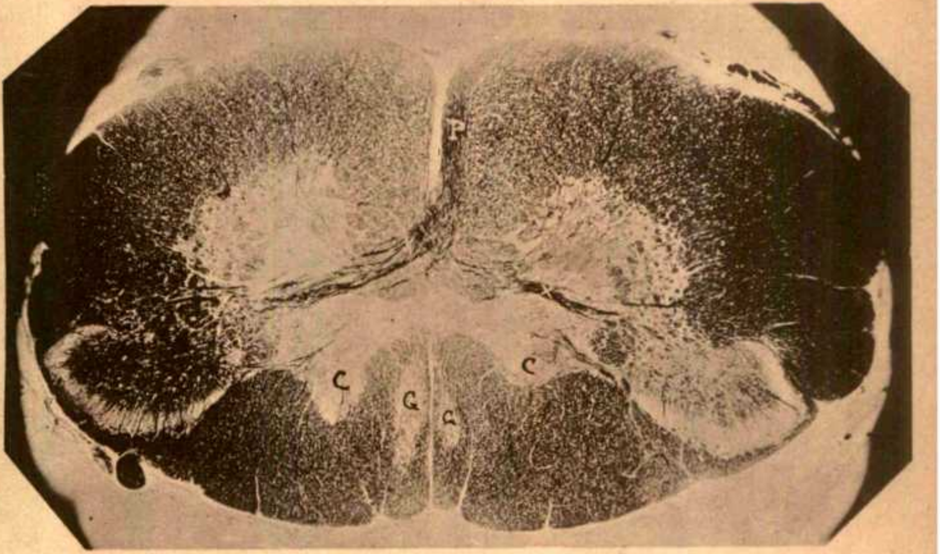

FIG. 8.

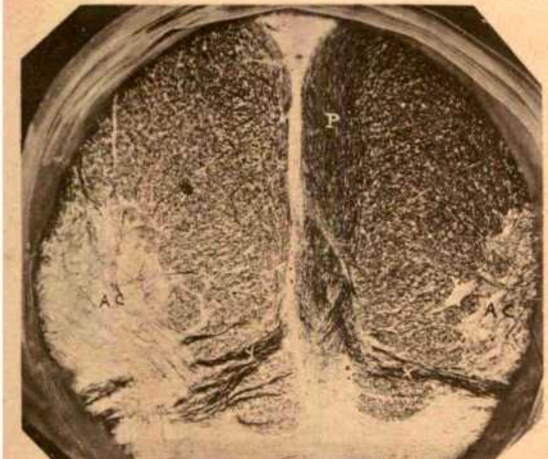

FIG. 9.

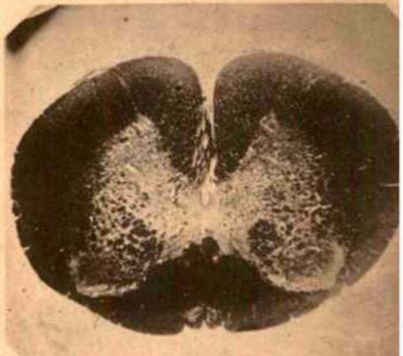

FIG. 10.

influence on the opposite half of the cerebrum has been strikingly demonstrated by certain of my experiments,[1] which leave little doubt that one lateral half of the cerebellum exercises a control on the cortical cells of the opposite cerebral hemisphere.

In the case we are at present considering, although there is very great deficiency of the right lateral lobe of the cerebellum, there is no sign of atrophy or other defect of the left cerebral hemisphere. On the contrary, it is the right cerebral hemisphere which is slightly smaller than its fellow, a condition which is probably part of the general defect on the right side of the central nervous system, and consequent on some common cause, with these other defects, and not in any way specially associated with, or secondary to, the defective development of the right lateral lobe of the cerebellum.

As we are considering the question of crossed relationships, it will be as well for us to inquire into the probable significance of the fact that the posterior column nuclei are smaller on the left side, which, with the exception of the inferior olive, is otherwise quite normal. That these nuclei should be smaller on the left side is only what we are led to expect from the complete absence of the fillet on the right side, for the relations of the fillet to the opposite posterior column nuclei is well recognised. Indeed, it is a matter for surprise that with apparently so complete an absence of fillet fibres on the right side the nuclei of the funiculus gracilis and funiculus cuneatus should not have been considerably smaller than they are; especially when we remember that nothing abnormal could be detected in the basal ganglia.

It only remains for us to consider the defects met with in the pyramidal system, and their probable significance. The internal capsule being a little smaller on the right side than on the left is of course in keeping with the fact that the right hemisphere generally is a little smaller than the left. The size of the right crus cerebri is also in keeping with this;

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[1] *Phil. Trans. Roy. Soc.*, 1894.

but neither the size of the capsule nor of the crus prepares us for the complete absence of the right pyramid at a lower level. With such a complete absence of the pyramid on the right side, one would have been justified in looking for a greater diminution in the size of the crus and internal capsule, and certainly also in the cortex of the right cerebral hemisphere.

It is customary to designate those fibres which occupy the ventral aspect of the mesencephalon, in the region of the posterior corpora quadrigemina, pyramidal fibres; but that this is probably not strictly correct is rendered likely from the condition met with in this animal. We have seen that in the region of the corpora quadrigemina there is a distinct bundle of fibres occupying the region of what is generally looked on as pyramid, on the right side, though the bundle is very much smaller than the corresponding one on the left side. It is difficult to see how, if this bundle of fibres on the right side is wholly made up of pyramidal fibres, there should not be a trace of them to be found in the region of the pyramid at a slightly lower level. Boyce[1] has shown that pyramidal fibres leave the pyramids at all levels in their course, but the number of fibres which thus leave the pyramids under normal conditions are not sufficient to account for the entire disappearance of so considerable a bundle in so short a vertical space. It seems to me far more reasonable to suppose that the fibres forming the bundle in question in the posterior quadrigeminal region are not pyramidal fibres at all, but are of a totally different nature. If this be so, then it appears to me that it would be far more correct to call the bundle at present called pyramid, in the region of the posterior corpora quadrigemina, crural fibres, for as in the crus, so here, there are other fibres in addition to the pyramidal ones. I am thus inclined to believe that under normal circumstances there are certain other fibres contained in the bundle in question together with the pyramidal fibres, and that in this animal these fibres alone exist, while there are no pyramidal fibres contained in the bundle.

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[1] Boyce, *Proc. Roy. Soc.*, 1894.

At the level where the anterior pyramids decussate there are two points which call for our consideration. In the first place we have seen that although the pyramid is completely absent on the right side at certain higher levels we have in this region certain fibres occupying the position of pyramidal fibres, indistinguishable from such fibres, and behaving like pyramidal fibres in many respects, for while some go to the anterior horns, others pass to the crossed pyramidal tract of the same side, and some to this tract on the opposite side. It is useless to speculate on the possible source from which these fibres are derived, but their presence points to the probability that just as fibres leave the pyramidal system at different levels, so fibres are added to it at certain levels. I see no other way of explaining the existence of these fibres. If this be the true explanation of their presence, it is clear that the generally accepted belief that all the pyramidal fibres are derived from the cerebral cortex, would require modification. The other point of interest with regard to the pyramidal fibres at the decussation is the fact that while the bulk of the fibres contained in the left or normal pyramid cross to the opposite crossed pyramidal tract, a considerable number pass to the crossed pyramidal tract of its own side. This state of affairs entirely conforms with the results obtained by Mellus[1] and Sherrington[2] who on extirpation of limited foci of the cortex of one cerebral hemisphere of monkeys found that at the decussation of the anterior pyramids the majority of the degenerated fibres passed across to the region of the opposite crossed pyramidal tract, but that a few degenerated fibres passed to the crossed pyramidal tract of the same side as the hemisphere from which the portion of cortex was removed. In my animal the proportion of fibres passing to the crossed pyramidal tract on the same side, that is, on the side of the normal pyramid, were considerably in excess of the proportion met with by Mellus[3] in monkeys. Possibly this is an attempt to compensate for the absence of the pyramid on the right side at a higher level and the consequent meagre supply of pyramidal fibres to the left crossed pyramidal tract. This effort at compensation on the part of the normal pyramid accounts in great measure, no doubt, for the comparative symmetry of the spinal cord in the region of the crossed pyramidal tract; but whether it wholly accounts for it may be open to question.

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[1] Mellus, *Proc. Roy. Soc.*, 1894.

[2] Sherrington, *Lancet*, 1894.

[3] *Loc. cit.*

DESCRIPTION OF FIGURES.

FIG. 1.—Represents the cerebrum and cerebellum of a cat, and is intended to show that the whole cerebellum is smaller than normal but that the right lateral lobe is much smaller than the left.

R. = Right. L. = Left.

FIG. 2.—Is from a transverse section in the mesencephalon, and demonstrates that while the optic tracts are comparatively equal in size the right crus cerebri is considerably smaller than the left.

C = Crura cerebri. O. T. = Optic tracts.

FIG. 3.—Indicates the appearances met with on transverse section through the region of the posterior corpora quadrigemina. The right corpus quadrigeminum is obviously much smaller than the left, and there are far fewer crural fibres on the right side, on which side there is almost a complete absence of the fillet and superior cerebellar peduncle.

C. = Crural fibres.
F. = Left fillet.
S. = Left superior cerebellar peduncle.
D. V. = Descending root of fifth nerve.
R. = Right. L. = Left.

FIG. 4.—A section through the decussation of the fourth nerves in the roof of the aqueduct of Sylvius, and shows that the right nerve is much smaller than the left.

A. S. = Aqueduct of Sylvius. IV. = Fourth nerves.

FIG. 5.—Represents a transverse section through the lower part of the pons; the most marked feature at which level is the entire absence of the pyramid on the right side.

P. = Left pyramid.
A. V. = Ascending root of fifth nerve.
O. = Superior olive.
VI. = Sixth nerve.
VII. = Seventh nerves, longitudinally and transversely divided.
R. = Right. L. = Left.

FIG. 6.—Is from a transverse section a little below the level from which fig. 5 was taken, and in addition to the absence of the right pyramid, it is intended to show the insignificance of the right restiform body as compared with the left.

R. = Restiform bodies.
V. = Ascending root of fifth nerves.
O. = Superior olives.
P. = Left pyramid.
R′ = Right. L. = Left.

FIG. 7.—Is a section of the medulla oblongata, made transversely through the level of the inferior olive, which is well marked on the right side and extremely diminutive on the left side. The pyramid and lateral medullary tract are absent on the right side.

G. = Funiculus gracilis.
C. = Funiculus cuneatus.
S. G. = Substantia gelatinosa.
L. M. T. = Lateral medullary tract.
P. = Pyramid.
O. = Inferior olive.

FIG. 8.—Is from a transverse section through the decussation of the pyramids, and shows that while the bulk of fibres from the intact pyramid pass to the opposite crossed pyramidal tract, some pass to the crossed pyramidal tract of the same side.

P. = Left pyramid.
X. = Fibres going from the left pyramid to the crossed pyramidal tract on the same side.
G. = Funiculus gracilis.
C. = Funiculus cuneatus.

FIG. 9.—Is from the same level as the last, but more highly magnified.

P. = Left pyramid.
A. C. = Anterior horn.
X. = Fibres from pyramid to left crossed pyramidal tract.
T. = Fibres from pyramid to right crossed pyramidal tract.
Z. = Fibres in position of right pyramid.

FIG. 10.—Represents a transverse section through the upper part of the cervical cord and shows that the two lateral halves of the cord are as nearly as possible equal.

NOTES OF A CASE OF DUAL BRAIN ACTION.

BY LEWIS C. BRUCE, M.B.

Assistant-Physician, Royal Asylum, Edinburgh, late Assistant Medical Officer, Derby Borough Asylum.

THOUGH the study of man and the brain of man has for long ages attracted philosophers and thinkers of every cultured race, and though Plutarch informs us that the physician Alcmæon regarded the brain as the common meeting-place of the senses, it was not till the year 1840 that Sir H. Holland, in this country, brought forward the subject of the brain as a double organ.

Since then many writers have directed their attention to the subject, and of late years hypnotism has been employed in an endeavour to penetrate still further into the mysteries with which the problem is beset.

There appears to be considerable ambiguity among various writers as to the precise meaning of the term "dual brain action." In this paper I use the term as describing an individual who appears to have two separate and distinct states of consciousness, and in whom the right and left brain alternately exert a preponderating influence over the motor functions.

The following case presents points of special interest both in psychology and physiology.

H. P., a Welshman, aged 47, by occupation a sailor, was admitted to the Derby Borough Asylum, on October 25, 1892, as a boarder from a neighbouring asylum.

Previous history very defective. He had been insane for fifteen years. His early symptoms were those of melancholia.

Latterly he was a noisy, distructive dement and required special clothing and bedding.

On admission his appearance was not prepossessing. He was small, bent but wiry, face wizened, features irregular, hair red and coarse, expression one of extreme degradation and cunning.

Mentally he was absolutely demented and did not understand a word that was said to him. He frequently gave vent to a jabbering sound which was not English, but may have been Welsh. On November 20, 1892, his manner was completely altered; he was restless, talkative, destructive and mischievous. He expressed himself in English and understood what was said to him.

This condition continued until the middle of December, when he gradually relapsed into the apathetic demented condition.

At irregular intervals between this date and November, 1893, he had periods of restless, destructive excitement.

As his language, in the two mental conditions, was the most striking and obvious of the phenomena observed, I speak of them to save repetition, as the English stage and the Welsh stage.

Observations and notes made at regular intervals were somewhat misleading. I therefore thought it advisable to observe his condition continuously for three months, in the hope of being able to throw light on the contradictory phenomena observed.

These observations form the basis of the succeeding remarks.

November 30.—He was sitting huddled up in his chair, paying no attention to his surroundings. I asked him if he would like a penny. He apparently did not understand the question and looked up for a moment with a vacant expression. He was shown and offered a penny, but did not appear to recognise the coin, and only after it had been offered to him for a considerable time did he slowly put out his left hand and take it, but with no sign of eagerness. He was shown and offered tobacco, but looked at the gift without interest and did not wish to take it. At his meals he used the left hand exclusively for conveying food to his mouth. He spoke in Welsh.

December 30.—He was rather more active, spoke more and looked brighter. The right hand was moved more frequently.

January 2.—Understood the word tobacco. Was right-or left-handed as most convenient. Was speaking Welsh with an occasional English word.

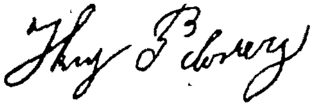
January 5.—Was speaking complete English sentences, but occasionally used a Welsh word. Understood English and was ambidextrous.

January 6.—He ate his dinner to-day, using the spoon in the right hand. He was restless, thievish and inclined to be destructive to clothing and bedding.

January 8.—Relapsed to the Welsh stage, was quite demented and used the left hand exclusively.

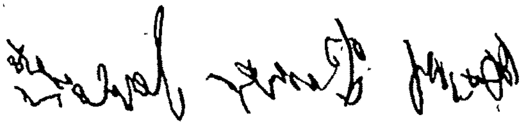
January 11.—Since last note he has been in an intermediate condition between the Welsh and English stages. When speaking, he employed both English and Welsh words. He was ambidextrous, *e.g.*, he drank tea carrying the cup with equal facility in either the right or left hand.

January 20.—He has again relapsed into the Welsh stage.



SPECIMEN No. 1.

January 29.—Written with right hand from left to right. (Intended for Hugh Parry.)



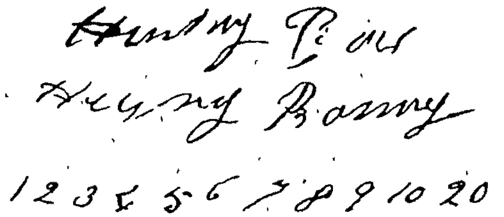
SPECIMEN No. 2.

January 29.—Written with left hand from right to left. (Intended for Hugh Parry.)

January 24.—He woke up to sudden life to-day, was ambidextrous and spoke in both Welsh and English.

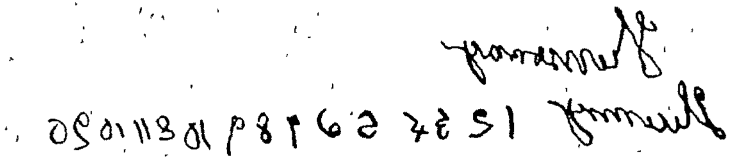
January 27.—Has again become demented and completely left-handed. A Welsh patient interpreted for me to-day, but made little of him. My interpreter said that the jargon H. P. spoke was unintelligible and that he could only detect a Welsh word here and there. After many questions had been asked, H. P. replied gruffly, in Welsh, "I will answer no more!"

January 29.—10 a.m. He was slightly restless, spoke an occasional English word in his chattering. He was distinctly left-handed, but the right hand, which in the Welsh stage is always in the trouser pocket, was being used slightly. 12 o'clock noon. He employed both hands when eating his dinner and was rather awkward in movements with the left. He understood English but replied in a mixture of Welsh and English words. He was asked to write his name. He took the pencil at once in the right hand but wrote awkwardly. (Specimen of hand-writing No. 1.) It was suggested to him that he ought to write with his left hand. He did so and wrote with ease, but the writing was what is commonly known as mirror writing, that is to say, he wrote from right to left, making his letters backwards. (Specimen of hand-writing No. 2.) 6 p.m. He was completely English and right-handed. He wrote readily with the right hand but needed great pressure before he would use the left, and his writing with the left hand was now much more awkward than it had been at mid-day. He recognised coins and approximately stated their value. He asked for tobacco and two or three times tried to steal both coins and tobacco from my pockets. He readily understood when spoken to in Welsh, but apparently preferred to speak English.


Henry Ramy
1 2 3 4 5 6 7 8 9 10 20

SPECIMEN No. 3.

January 30.—Written with right hand from right to left.

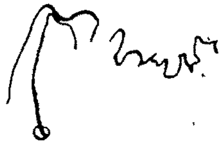


SPECIMEN No. 4.

January 30.—Written with left hand from right to left.

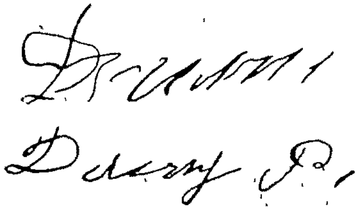
January 30.—In English stage. He was very restless, destructive and constantly stealing from his fellow patients. He employed the right hand almost exclusively, even at his meals. A specimen of his writing was obtained and his awkwardness in his use of the left hand again observed. (Specimens of hand-writing Nos. 3 and 4.)

January 31.—In the Welsh condition and completely left-handed. After great difficulty a specimen of his writing was obtained. He took the pencil in the left hand and wrote a scrawl, of which the first letter resembles a P. He likewise scribbled at random, all his movements being from left to right. (Specimen of hand-writing No. 5.) The pencil was removed from his left hand and put into his right but he promptly transferred it back to the left. After this manœuvre had been repeated many times he suddenly, and in an impulsive manner, wrote with the right hand, writing from left to right. The only letter at all legible was a P and that he wrote backwards. (Specimen of hand-writing No. 6.) A Welsh patient interpreted for me during this investigation.



SPECIMEN No. 5.

January 31.—Written with left hand from left to right.



SPECIMEN No. 6.

January 31.—Written with right hand from left to right.

With a view to ascertaining his condition during the night and when awakened from sleep, he was observed frequently by the night attendants.

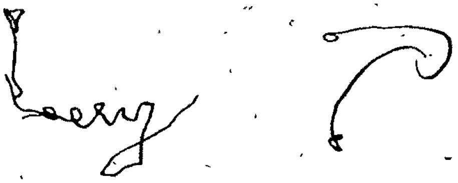
February 1.—He was in the English stage all day. 9 p.m. Awakened from sleep, he was in the Welsh stage and left-handed. 12 o'clock midnight, in Welsh stage. At 1, 2, 3, 4, 5 and 6 a.m. February 2, he spoke English.

February 2.—Was in the English stage all day. 9 p.m. When awakened from sleep he was completely left-handed and speaking Welsh. At 12 o'clock midnight, 3 and 6 a.m., February 3, still in Welsh stage, but at 6.30 a.m. was back to English stage.

February 3.—He was restless, destructive and in the English stage till bedtime. Awakened at 9 and 11 p.m., 12 o'clock midnight, 2, 3, 4, 5 and 6 a.m., February 4; on each occasion spoke English.

February 4.—He was more than usually dirty, restless and mischievous. Spoke English and was completely right-handed. He wrote again to-day, but experienced great difficulty in using the left hand.

February 5.—All last night he was in the English stage. This morning at 10 a.m., he was less restless and appeared slightly confused. By 12 o'clock noon he had passed into the Welsh stage. A specimen of writing was obtained after much trouble, but he resolutely refused to write with the right hand at all. (Specimen of hand-writing No. 7.)



SPECIMEN No. 7.

February 5.—Written with left hand from left to right.

He was under observation all night. At 8 and 9 p.m. was in the Welsh stage. At 11 p.m. and 12 o'clock midnight, he spoke in a mixture of Welsh and English.

February 6.—1 a.m. Spoke in English. 2 a.m. Spoke in Welsh. 3 a.m. Spoke in both Welsh and English. 4 a.m. Spoke in English. 5 a.m. Spoke a mixture of Welsh and English. 6 a.m. Spoke in English. For the rest of the day was in the Welsh stage. At 9 p.m., when wakened from sleep, was in the English stage and continued in this stage all night.

February 7.—In the English stage.

February 14.—Since last note, has been in the English stage. To-day he is speaking both Welsh and English and uses the left hand more than the right.

February 15.—Was in the English stage all day.

February 18.—At dinner-time he passed suddenly into the Welsh stage.

February 19.—Continued in the Welsh stage all day, but by 6 p.m. he was in the English stage and right-handed.

February 21.—Passed into the Welsh stage.

February 22.—Again returned to the English condition.

March 1.—Has continued in the English stage since last note.

It would appear that in this case, either cerebral hemisphere was capable of independent action, more especially with regard to mental processes, the dual action being confirmed by the invariable change from right-handedness to left-handedness or *vice versa*, which coincided with the alternating English and Welsh stages.

When in the English stage, right-handed and presumably using the left cerebral hemisphere, he is the subject of chronic mania. He speaks in English but understands and will converse in Welsh. He is restless, destructive, thievish, and constantly playing practical jokes on his fellow patients. He exhibits a fair amount of intelligence. He writes, draws pictures of ships, and relates incidents in his past life. He is bold and fearless in manner. He recognises the doctors and attendents. His memory is, however, a blank to anything that occurred during the Welsh stage. For instance, he got his arm burnt and blistered once when in the Welsh stage. He passed into the English stage a few days after the accident, but could give no account of how he came by the injury.

On the other hand, he remembers clearly things he has noticed in previous English periods; for example, he remembered the mottoes of the Christmas ward decorations a year afterwards, repeated them correctly and pointed out the positions they had occupied.

His circulation is good, his appetite increased, and his bowels move regularly. He is fond of bathing and would roll and splash in the bath for an hour if allowed.

With regard to the observations made on him in the English stage, his writing is worth noting. He writes by preference with the right hand, his letters are fairly legible, and he writes from left to right. If asked to do so, he uses the left hand, but now produces mirror writing, traversing the paper from right to left.

Then, looking at the results obtained by a study of the special senses :

Sight is active, he can distinguish different coins. He recognises tobacco, calls twist, twist, and cut tobacco, shag. He readily distinguishes between the primary colours, red, yellow and blue, and can assort different tints of these colours into their respective classes.

Hearing is acute; he readily understands English and Welsh. He is pleased with the sound of a tuning fork, but appears to have no musical sense.

Taste.—Detected salt and sugar. Tasted and did not like lin. camph. co. or assafœtida.

Smell.—Called lin. camph. co. a nice smell, and assafœtida castor oil.

Touch.—Sensibility to heat, pain and touch unimpaired. He is regardless of the severest weather, and will walk about with his chest exposed in a biting wind.

When in the Welsh stage, left-handed and presumably using the right cerebral hemisphere, he is the subject of dementia.

His speech is almost unintelligible; I have got Welsh patients to act as interpreters; they tell me that they cannot understand much of what he says, but what they do understand is spoken in the Welsh language. He appears to understand Welsh, however, as occasionally he will answer a question. He has now no idea of English, does not understand the simplest remarks, but stares vacantly at the questioner as if endeavouring to discern his meaning.

His mental and physical conditions are the reverse of what they were in the English stage.

He sits doubled up in a chair for hours and never offers to move until meal times, when he requires to be started on his way to the dining hall.

It is impossible to estimate his education or memory.

He is now shy and suspicious, does not recognise the doctors or attendants, and appears to be constantly on the look out for unseen danger.

His circulation is weak, his extremities livid and his legs often become œdematous.

He suffers from constipation and passes small quantities of urine at long intervals.

He dislikes bathing and will not stay in the bath without persuasion.

Specimens of his hand-writing are now obtained with difficulty. He always uses the left hand and traverses the paper from left to right. On one occasion he used the right hand, after much persuasion; he traversed the paper from left to right, but made his capital letters backwards.

His special senses are unimpaired, but he is no longer able to analyse and appreciate the crude sensations received.

Sight.—Coins he merely looks at but does not try to possess himself of them. Tobacco is likewise not recognised. Colours he only glances at, and when asked in Welsh to sort out the three primary colours he is quite unable to do so.

Hearing.—He does not understand English, but occasionally gives evidence of understanding Welsh. He is alarmed at the sound of a tuning-fork and tries to avoid having the instrument near him.

Taste.—Did not prefer sugar to salt. Tasted, but showed no like or dislike for lin. camph. co. or assafœtida.

Smell.—The strongest and most pungent odours produced no effect, and he tried to drink lin. camph. co. and assafœtida after smelling the bottles containing these drugs.

Touch.—Sensibility to heat, pain and touch unimpaired. He appears to feel cold weather, and constantly pulls his jacket more firmly round him.

The presence of distinct bodily symptoms peculiar to the alternating mental phases is one of the most noticeable features of the case. No previously reported case, which I have read, exhibited such symptoms in so marked a manner.

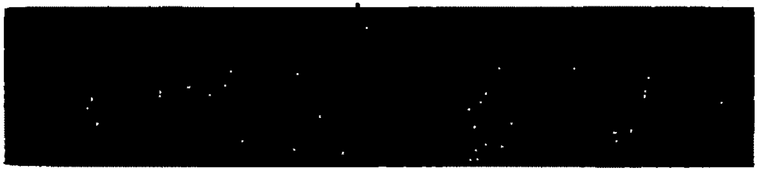
Occasionally, when changing from the Welsh to the English stage, or the reverse, this patient passes through an intermediate condition, in which he is ambidextrous, speaks a mixture of Welsh and English and understands both languages.

This intermediate stage is often absent, or so short as to pass unnoticed; the patient suddenly waking up to life and activity, or becoming suddenly demented.

These sudden changes generally occur after a meal or after a bath.

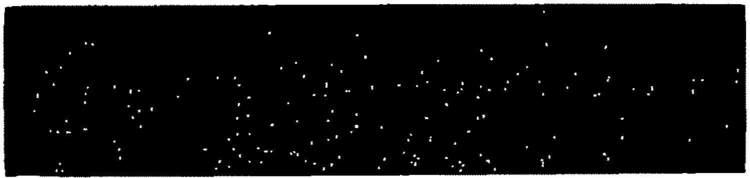
As I believe some circulatory change to be the direct cause of the transition from one stage to the other, I took several sphygmographic tracings of the radial pulse.

The tracing obtained during the English stage presents the following characters:—Cardiac percussion stroke of fair length and slightly oblique. The apex and predicrotic wave tend to form a rounded plateau. The line of descent, moderately sustained, presents traces of the aortic notch and dicrotic wave. The general characters of the tracing denote a pulse of high tension.



Typical example of pulse tracing in English stage. Pulse 100, pressure 5 oz.

In the Welsh stage it was almost impossible to obtain a tracing, as the patient regarded the sphygmograph with horror and fear. After several attempts two tracings were obtained which closely resemble each other, and I give these as characteristic of the pulse in this stage.



Examples of pulse tracings in Welsh stage. Pulse 105, pressure 5 oz.
Pulse 110, pressure 5 oz.

The cardiac percussion stroke is short and almost vertical. The apex is acute, while the line of descent is oblique, and presents the aortic notch and dicrotic waves. A pulse of lower tension and less volume than in the English stage.

I have endeavoured to produce the different stages artificially, by subjecting the patient to baths of various temperatures, and though, to the finger, the character of the pulse was altered, no change was produced in the patient's mental condition.

From these observations it would appear that in this case, the cerebral hemispheres are capable of individual mental action, and that the mentally active cerebrum has a preponderating influence over the control of the motor functions, the patient living two separate existences during the two stages through which he passes; the mental impressions received during each of these separate existences being recorded in one cerebral hemisphere only.

If this is not so, how can one account for the patient's ignorance of events which have happened to him in the Welsh stage when he passes into the English condition? Or his suspicion and distrust of attendants and doctors, ignorance of familar and much coveted objects, as money and tobacco, when in the Welsh stage, whereas, in the English stage, he recognises and is very friendly with the staff, while the sight of money or tobacco is sufficient to bring him running the length of the ward.

A comparison of the mental power of either cerebral hemisphere places the right at a much lower level than the left. Is this due to the unequal ravages of disease or to the unequal development of education?

Judging this case by its own characteristic symptoms, one inclines to the belief that even in health each cerebrum must have acted independently, and received external impressions separately; that on the advent of mental disease, each cerebral hemisphere still maintained its individuality, the left exhibiting symptoms of mania, the right those of melancholia, and that the disease has advanced more rapidly in the right brain, which now exhibits symptoms of dementia. Or it is suggested, that only one lobe of the cerebrum, the left, is educated, and takes the greater part in mentalisation; that the right half, though functionally active with regard to motion, sensation and the lower reflexes, has little education or power of mental action, except that which it derives from inheritance; that the centres in connection with sight, smell, taste and hearing are equally developed in both hemispheres, but only one side, the educated one, is able to translate, analyse and mentally appreciate the crude sensations received. (Dr. Macphail has kindly given me permission to publish this case.)

MENTAL STUPOR AS A PATHOLOGICAL ENTITY.

BY JAMES R. WHITWELL, M.B.
Medical Officer, West Riding Asylum, Menston.

Amongst the numerous admissions to our hospitals for mental disease, there is a comparatively small group which may be justly regarded as comprising cases of primary stupor, cases in which the main and characteristic feature is bluntness of mental and nervous reaction, preceded though it may be by a shorter or longer stage of irritability and loss of mental balance. This group has been defined by Dr. Clouston[1] as "a morbid condition in which there is mental and nervous lethargy and torpor, in which impressions on the nerves produce little or no outward present effect, in which the faculty of attention is or seems to be paralysed, in which there is no sign of originating mental power, in which the voluntary motions are almost suspended for want of convolutional stimulus, but where the patients usually retain the power of standing, walking, masticating and swallowing."

From a clinical and pathological study of a series of cases of so-called mental stupor I have endeavoured to point out[2] that there is some considerable weight of evidence in favour of the view that these cases or certain of them, may be due to a want of normal proportionate development in the circulatory and nervous systems; that want of the due ratio in time of development of these two systems leads to nerve cell malnutrition or dystrophoneurosis, and consequent imperfect mental action; that, in fact, while the brain reaches the degree of development, normal to the age, sex and physique, the blood vessels, and frequently also the heart, remains in its puerile condition, the aorta and other blood vessels throughout the body, therefore, showing a condition of congenital narrowness, associated in some cases with a congenitally small heart. If the cardio-vascular condition be only temporary and under proper stimuli continue to undergo its development, finally the normal vasculo-cerebral proportion is brought about, and the patient recovers from his mental stupor. If, however, a condition of permanent hypoplasia obtain, the case sinks into dementia, and is either carried off by some inter-current acute disease or, as is frequently the case, develops phthisis.

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[1] "Mental Diseases," third edition.
[2] *Journ. Mental Science*, Oct., 1889.

This theory as to the causation of mental stupor was based on the following points :—

(1) Diminution in the calibre of the basal cerebral vessels or aorta or both, I have shown to be actually present in certain cases, not unfrequently associated with a general hypoplasia of the circulatory system.[1]

(2) The experimental study of the relation of cortical reaction to stimulus, in proportion to blood supply, shows an exact parallel to cases of mental stupor.[2]

(3) The clinical mental symptoms are, from our present knowledge of cerebral action, and from the parallel in other organs, fully in accordance with the possibilities of such a lesion.

(4) The clinical physical examination frequently reveals a heart lesion known to occur in cases of congenital vascular narrowness.

(5) The sphygmograph shows a high tension pulse indicating peripheral resistance.

(6) The change of tension in the pulse coincident with the mental change can be traced in certain cases of intermittent stupor.

(7) The action of nitrites leading to peripheral arteriolar

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[1] *Journ. Mental Science*, 1889.

[2] Horsley and Spencer, *Brit. Med. Journ.*, 1889.

dilatation is well known as a method of producing some temporary mental improvement in some of these cases.

Two other cases of mental stupor which have died recently under my care also give some evidence in favour of this being a possibly correct pathological cause of stupor; if so, however, further experience must decide how general is its causative influence, or whether, on the other hand, it will become necessary to divide our existing clinical field into separate pathological compartments.

Case 1.—H. C., printer, aged 21, admitted October 27, 1893. Said to have been normal mentally till two years previously, when he received a shock it is said from a friend of his being drowned while out bathing with him. Since this period he has appeared to take less interest in his work, to be more irritable and ill-tempered, and readily got into a passion on slight cause. Later he became more and more stupid, taking little or no interest in or even notice of things around him, and requiring finally everything to be done for him. When examined by the doctor previous to admission he took no notice whatever of him or of any of his own relatives present; he had a vacant, unintelligent expression, did not do anything he was told to do and sometimes laughed immoderately without cause. On admission, the patient was anæmic, and in a state of typical mental stupor of a moderate degree, *i.e.*, he would stand still in one position for hours, but when started would walk a little way, and when the action was commenced passively would feed himself. This stupor deepened somewhat and a slight tendency to cataleptoid rigidity obtained and speech was only obtainable by the additional stimulus of the interrupted current. On one occasion he made an impulsive attack on an attendant, and then immediately relapsed into his stupid condition again. Sphygmograms of the pulse shew high tension; the aortic second sound was accentuated. This mental condition remained practically unchanged during his residence in the asylum, and in five months he developed acute colitis and died in ten days.

An aunt insane and mother neurotic.

Case 2.—A. H., shoemaker, aged 22, admitted November 14, 1881. About three months previous to admission he began to be a little strange, and unable to do his work so well as formerly, apparently owing to inattention. He answered questions in a strange, "contradictory" manner, and was said to be always "meditating" and "studying." Later he became more stupid and hardly ever spoke unless first addressed; he became steadily worse and took no notice of anything, facial expression became vacant and he sometimes laughed suddenly without apparent cause. This condition of mental stupor deepened until the patient took no notice of his surroundings, stood about for hours in the same attitude, was wet and dirty in his habits, rarely spoke when spoken to, or at times muttered unintelligibly, and required starting in most of his movements. Gradually dementia came on, and phthisis terminated a case of twelve years' standing. Heredity unascertainable.

TABLE I.

Name.	Sex.	Age.	Aortic dia	Ment. Cond.	Death by	Heart weight.	Brain weight	Duration.
H. C.	M.	23	16 mm.	Stupor	Enteritis	298 grm.	1,555 grm.	5 months
A. H.	M.	35	19 ,,	Stupor	Phthisis	155 ,,	1,140 ,,	12 years
R. D.	F.	21	19.2 ,,	Idiocy	Phthisis.	125 ,,	1,195 ,,	17 ,,
F. S.	M.	18	15 ,,	Idiocy	Pneumonia	160 ,,	1,115 ,,	18 ,,

TABLE II.

Orifice.	Males.			Females.		
	Max.	Min.	Average	Max.	Min.	Average
Aortic.	33·3	23	25·6	25·6	20·5	23

These, then, may be taken as typical cases of so-called stupor, each with an early stage of "irritable weakness," followed by a stage of apathy. In each the onset of the disease occurred about the age of 20; in one of the cases a distinctly neurotic history is present, and some causative influence may be possibly attributed to the shock received, if it be regarded as an "exhaustion of the medulla" producing a profound impression on the nervous and vascular systems.[1]

In both of these cases a congenitally small aorta was found. The two upper cases in table i. shows at a glance this condition, and also the weight of the heart and brain. The degree of stenosis present will be readily appreciated when a comparision is made with the table shewing the normal aortic diameters (reduced for convenience into millimetres) taken from Hamilton.[2]

The case of H. C. demonstrates the tendency of the heart to hypertrophy in the early young and otherwise healthy cases, and had it not been for the stepping in of an acute inter-current disease, it is possible that recovery may have occurred in this case. The basal cerebral vessels were not noticably small in either of these cases; the aortic diameters were, however, in both of these obviously well below the minimum female measurements, regarding Hamilton's extended and careful observations as standards. In four cases of mental stupor, however, that I have elsewhere[3] recorded, the basal vessels were diminished in calibre to a remarkable degree, in some instances far below the normal, in addition to the general hypoplasia of the circulatory system throughout the body. This puerile condition of the vessels is perpetuated in many idiots whose vascular system does not apparently undergo that slow post-pubertal evolution which occurs normally, and which is in all probability intimately associated with the incidence of the higher mental and bodily development, normally occurring about this time: this is shown in the two lower cases of the series in Table I. Vascular hypoplasia may even be regarded as a cause of idiocy in some cases, as in a case of microcephalism recorded by Jensen, the carotid canals were diminished in size by about one-fourth of the normal calibre of these apertures.[4]

Between puberty and complete manhood the brain increases in weight to the extent of about 19 per cent. of its total, and in addition, during this period, all a man's higher mental and bodily functions are developed; this, therefore, is a period of great nutritional demand in the nervous system; the circulatory system, however, is only under normal conditions developing but slowly after the period of puberty (this latter being a period of rapid increase in this system);[1] the result is that any delay in the rapidity of development of the vascular system must presumably detrimentally affect the nervous system, whose vascular supply is normally carried on under some difficulty. Now the effect of insufficient blood supply to the brain has been experimentally studied, and it can be shown that reaction to electrical stimuli is slower, and to produce the same result as when the blood supply is normal, it is necessary to increase the strength of the current,[2] just as in cases of stupor the additional stimulus of shaking, or the application of the interrupted current, will not unfrequently evoke a response, in answer to a question.

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[1] Groeningen, "Weber der shock," 1886.

[2] "Text-book of Pathology," vol. i.

[3] *Op. cit.*

[4] Quoted by Joseph, *Zeit. für psych.*, Bd. xxxvii., Heft 4.

I would here also record a most interesting case of intermittent stupor, which is still under observation, some details of whose pulse have appeared elsewhere.

B. W. H., clerk, aged 23, admitted July, 1889. First shewed mental symptoms at the age of 17, when he was confined in an asylum in Germany for over a year; later again he was in Netley Hospital for a similar condition; these two attacks his mother says were similar in all respects to the present one. On admission he was in a condition of profound stupor, standing in one position with head bent down and saliva dribbling from his mouth, takes no notice of anything going on around him, but with sufficient additional stimulus can be made to move a little. His limbs when placed in peculiar attitudes slowly change their positions in accordance with the action of gravity, but not the principle of comfort. This condition lasted for thirty hours, and then patient became bright, intelligent and talkative, interested in his surroundings, and fairly keen in work, games, and pleasure; this lucid period lasted twelve hours, the patient again lapsing into profound and typical stupor. This markedly intermittent condition, as may be seen from the accompanying hourly chart, continued till about the end of 1890, when dementia gradually came on, causing the mental condition to be never typically stupid (in the clinical sense) and never really lucid. Physically he is a fairly well-built and muscular man, and on admission his heart was found to be somewhat hypertrophied, and the aortic second sound accentuated. Patient's aunt, a cousin and a brother are or have been insane.

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[1] Beneke, "Die anat. Grundlagen der Constitutions-Anomalieen," 1878.

[2] Horsley and Spencer, *Op. cit.*

In this case considerable light was thrown upon the relative condition of the vascular system in the states of lucidity and stupidity by examination of the radial pulse with the sphygmograph, which, on a large number of occasions, showed exactly and invariably the same peculiarity. On one occasion it was possible to retain the sphygmograph *in situ* sufficiently long to observe the pulse condition (1) during stupor period; (2) during the intervening awakening period; and (3) during the lucid period. These tracings showed that during the period of stupidity, high tension, indicative of peripheral resistance was the invariable rule, and this completely relaxed, giving rise to distinct dicrotism on the supervention of lucidity, the intervening period showing a sphygmogram midway in character between these two.[1]

A great variety of medicinal agents were tried at different periods during the observation of this patient, interfering factors, such as diet, &c., being kept fixed, without, however, any noteworthy result. Under no treatment at all, he, as a rule, spent from 13 to 15 per cent. of his time "awake." The greatest change in this time occurred during the daily administration of bichloride of mercury; the period of lucidity then mounted to 28 per cent. of his time, a change which, however, can scarcely be regarded as of much importance.

The following propositions may now, perhaps, be fairly submitted in connection with the disease at present symptomatically classified as mental stupor.

(1) That there is at least one form of mental stupor associated with and probably due to a want of proportionate development between the vascular system, or part of it, and

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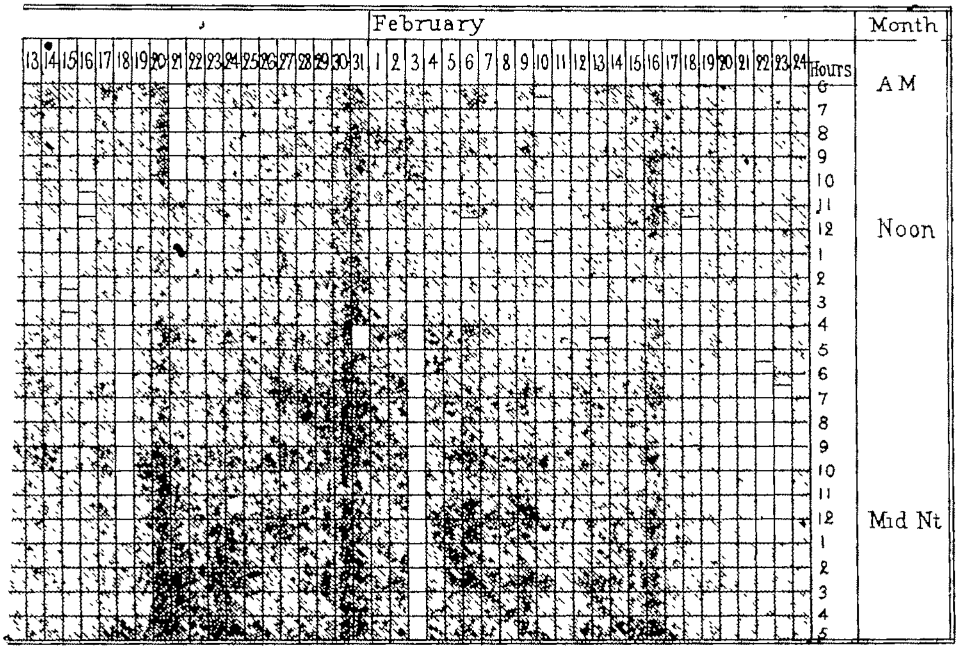
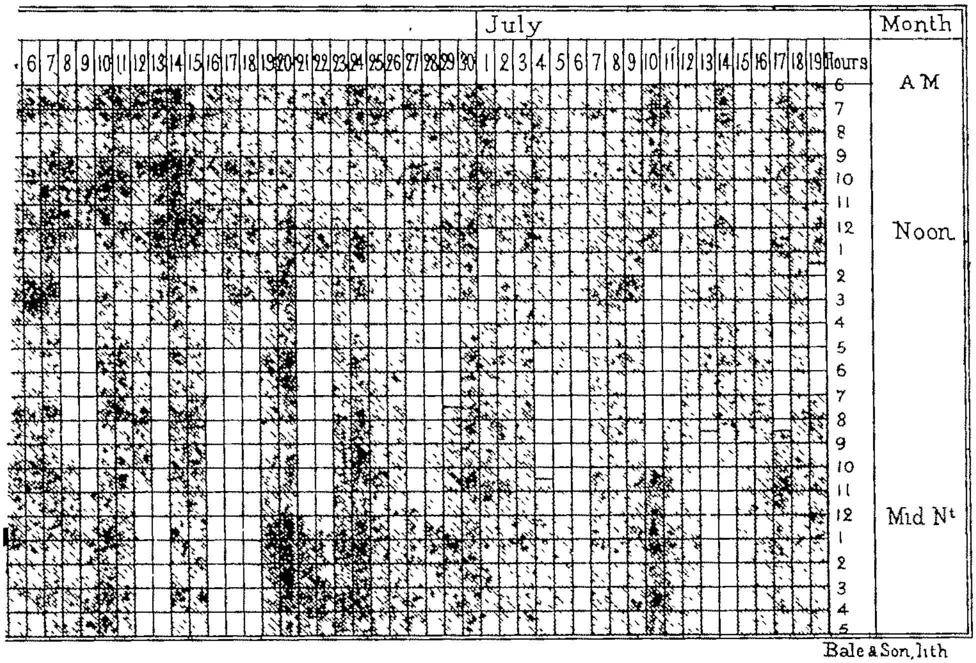
[1] *Lancet*, Oct. 17, 1891. "Stupor," "Dict. Psychol. Medicine," Tuke.





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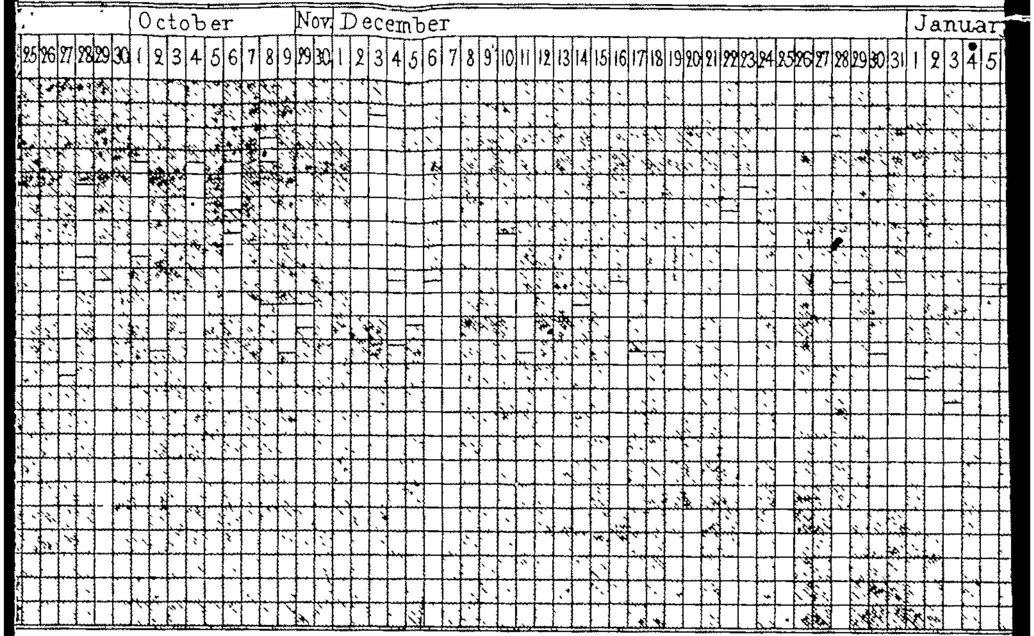
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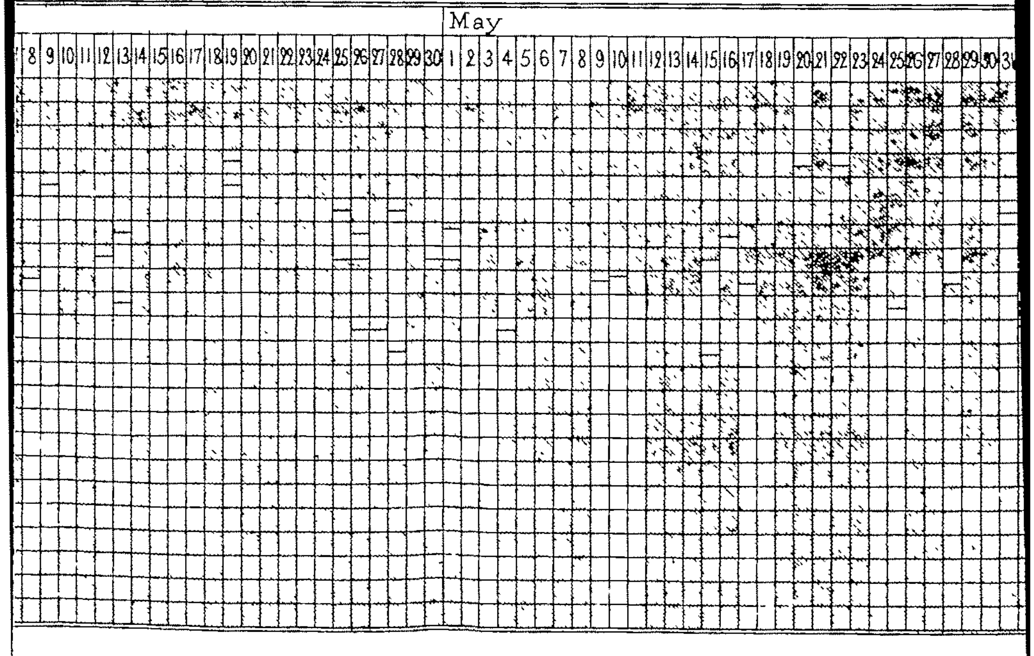
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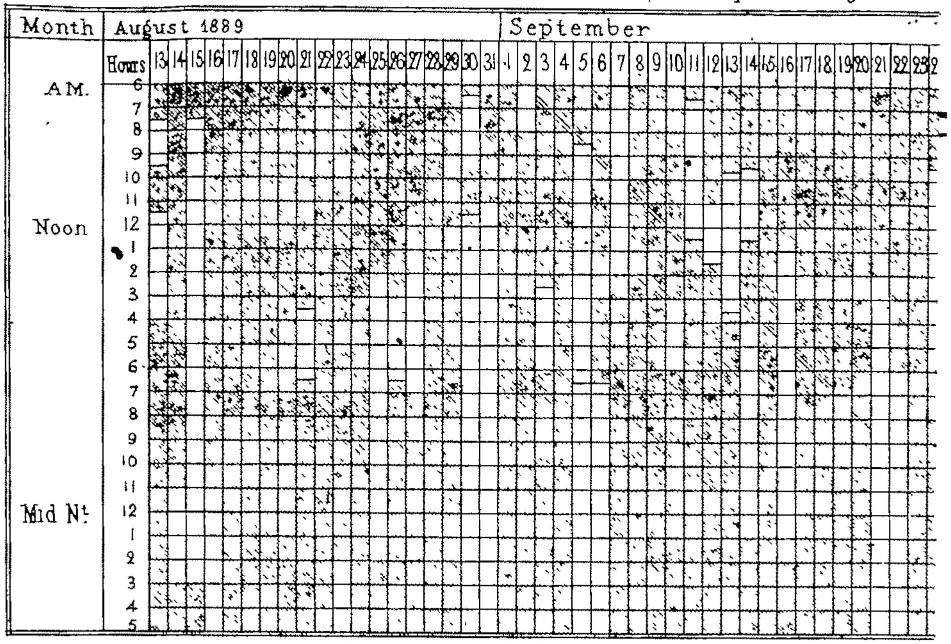
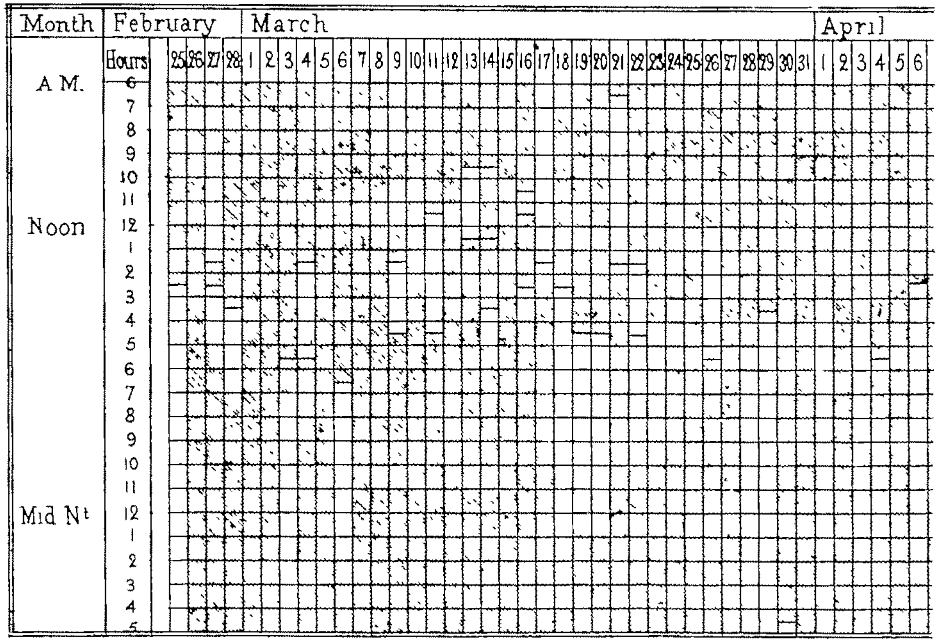
Case of BWH Intermittent stupor (*The open areas indicate periods of lucid*





the brain, and for this condition I have suggested the name "stenotic dystrophoneurosis" in order to differentiate it, if possible, by a name having a pathological significance.

(2) That in these cases either (*a*) the cerebral basal vessels, or (*β*) or the aorta and heart (omitting the question of compensatory hypertrophy), or (*γ*) all of these, are smaller than normal, having ceased developing at a stage of evolution about puberty or adolescence.

(3) That during life the heart tends to hypertrophy, when the patient is otherwise healthy, the aortic second sound to be accentuated and the pulse to be of high tension.

(4) That delayed cardio-vascular development about puberty and early adolescence may be associated with a form of mental stupor from which recovery is possible, with hypertrophy of the heart.

(5) That cessation of cardio-vascular development about this time may be associated with a form of mental stupor, from which recovery is impossible, and no hypertrophy of the heart is found *post-mortem*.

(6) That there is an intermittent form of stupor, caused by or associated with temporary spasm of the peripheral vessels during the period of mental stupor, this spasm relaxing during the period of lucidity.

CONTRIBUTION TO THE CLINICO-PATHOLOGICAL STUDY OF CEREBRAL LOCALISATION.

BY JAMES SHAW, M.D.

The following thirty cases came under my observation between June, 1879, and October, 1886, when I was medical officer of Haydock Lodge Asylum. Mental feebleness of various degrees was present in all of them. The other prominent symptoms and the coarse lesions are given without reference to their bearing on any particular theory of localisation or on the views of the opponents of all cerebral cortical localisation.

In sixteen cases there was hemiplegia or brachio-crural monoplegia.

Case A.—In one of the cases of hemiplegia there was anæsthesia of the right—that is, the paralysed side. The only lesions were:—(1) A focus of hæmorrhagic softening immediately under the cortex near the middle of the left second frontal convolution; (2) Softening (with membranous adhesion) of the cortex of the posterior third of the same convolution, of the ascending frontal in its upper third, of the ascending parietal in the upper half of its middle third; (3) Reddish discolouration of the lenticular nucleus, internal capsule, and cornu ammonis where cross-vertical sections passed through the softened cortex; (4) Softening of the right gyrus angularis.

Case B.—In a second case of hemiplegia (left) there was on admission and till death nine weeks after, contracture of the arm and leg, but no anæsthesia; the patient was restless and noisy at night, constantly shouting that her body was dead; there was an uncontrollable tendency to contract bed-sores. The lesions were:—(1) A brown-walled excavation three-quarters of an inch antero-posteriorly by a quarter laterally and one-eighth deep in the cortex joining the upper ends of the right ascending convolutions; (2) A superficial brown patch a quarter of an inch by one-eighth on the upper ramus of the superior frontal convolution, three-quarters of an inch anterior to the last lesion; (3) Two small brown specks on the inferior ramus of the superior frontal near its base and a similar speck near the commencement of its middle third (fig. 1).

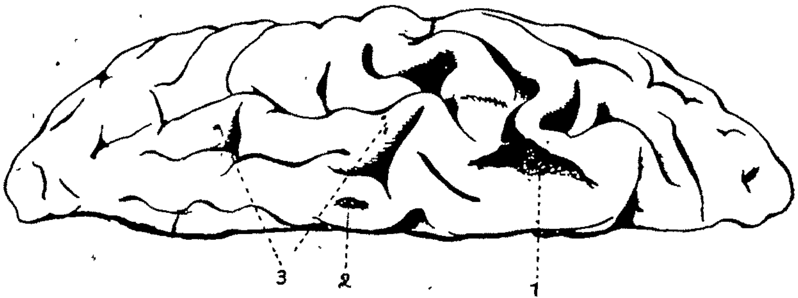


FIG. 1.—Case B.

Case C.—The third case of hemiplegia, also left-sided, was three months under observation; the vision of the left eye was defective; tendency to bed-sores; depression with suicidal attempts; capability of answering questions correctly so far as defective memory permitted; muscæ volitantes and tinnitus aurium. The lesions were:—(1) A large focus of yellow softening, as deep as the cortex, involving the whole of the right parietal lobe except the ascending convolution and the posterior half of the superior parietal; (2) Softening of the upper two-thirds of the right ascending frontal; (3) A focus a quarter of an inch in diameter in the right inferior frontal near its base; (4) A superficial focus three-quarters by half an inch in the orbital end of the latter connvolution; (5) Adhesion of the pia arachnoid to the occipital cortex of the same side.

Case D.—In the fourth case there was hemiplegia of the left side with frequently recurring convulsions of the same side and tremor in the intervals; the left pupil was widely dilated; pain in the right side of the head; patient, though dull, was at first able to converse. The only lesion was an abscess 1½in. in diameter in the right temporo-sphenoidal lobe close to the inferior surface with softening of the adjacent tissue.

Case E.—The fifth patient had a severe but transitory attack of hemiplegia (right-sided) some years prior to admission and suffered from mental aberration with hallucinations, amnesia, loquacity, incoherence, and insomnia; for a fortnight before death there was right brachial monoparesis. There were:—(1) Unusually large quantity of serous fluid; (2) Thickened arachnoid; (3) An old fibrous durhæmatoma; (4) Abnormal thinness and redness of cerebral cortex; (5) Depression and induration of inferior surface of right lobe of cerebellum.

Case F.—Another patient two years and a half prior to admission was seized suddenly with right hemiplegia and aphasia. The latter symptom, complete for a month, passed off gradually in the following five months. The hemiplegia persisted and was on admission accompanied by hemianæsthesia and hemianalgesia. The patient could walk, though lamely. He was unable to use his right arm, raise his right eyebrow, or to whistle; the right pupil was slightly the larger; the right knee-jerk was exaggerated; there was marked ankle clonus on the right side, none on the left. The patient could answer questions and converse, but his memory was defective both for recent and long past events, and there was emotional hyperæsthesia. Fourteen months after admission the facial paralysis and cutaneous anæsthesia had disappeared; the patient could move slightly his right arm and forearm, which latter was generally at right angles to the upper arm; he could extend his fingers but was unable to grasp; he could not move his right lower extremity, which was flexed and contracted, with the knee swollen; the left pupil was now slightly the larger; the patient could still move his left arm and leg freely; his mental condition was unchanged; occasionally he tried to sing, repeating, without time or tune, the words of a song. The right forearm was three-quarters of an inch less in circumference than the left, the right arm a quarter of an inch less than the left, the right thigh an inch and a half less than the left, the right leg (just above the ankle) a quarter of an inch less than its fellow. There was a tendency to bed-sores. The left cerebral hemisphere weighed only 473 grammes, the right 492·4, each hemisphere of the cerebellum 69·6, the pons and medulla 19·4, all free from membranes; 1,124 grammes or a little over 39½ ounces altogether. The only visible lesions were:—(1) Yellow softening, flattening, and marked diminution in size of the left nucleus caudatus; (2) Yellow softening with cortical loss of substance of the left gyrus angularis; (3) Slight yellow softening of the ventricular surface of the right nucleus caudatus; (4) Extensive degeneration of the left pyramidal fibres; (5) Slight of the right; (6) Several small lacunæ in the white substance of the left frontal lobe. (Patient aged 50.)

Case G.—In the seventh case there was on admission, and had been for six months previously, paralysis of the right arm and leg with general weakness; right pupil the larger; right knee-jerk the stronger; ankle clonus on both sides, more marked on the right; much mental weakness with visual and auditory hallucinations; patient restless, destructive, and dirty. Five months after admission and ten days before death, no clonus, no inequality of knee-jerks; pronounced right facio-lingual monoplegia was added to the previous brachio-crural, which became more accentuated, the hand, arm and leg being flexed but not contracted; the speech became thick and indistinct; cutaneous sensibility of the right side somewhat diminished especially on the foot. (1) The left nucleus caudatus was found softened, shrunken, and concave superiorly; (2) In the knee of the left internal capsule close to the nucleus caudatus and the corona radiata there was a recent clot one-twelfth of an inch broad and thick but longer antero-posteriorly; (3) A small lacuna in the right external capsule. The left cerebral hemisphere was 4½ grammes lighter than the right, and the right cerebellar 2 grammes 6 decigrammes lighter than its fellow; the whole brain denuded of membranes weighed only 33¼ ounces. The patient was aged about 70.

Case H.—The eighth patient began four years prior to admission to lose the memory for recent events, and six months before admission had an apoplectic seizure followed by complete paralysis of right arm and leg and slight of right face for 24 hours; then weakness of limbs for four or five months, gradually passing off. On, and for nine days after, admission no paralysis but staggering gait at times, restlessness, insomnia, mischievousness, aggressiveness. Then a slight apoplectiform attack with right hemiparesis, the right pupil being the larger. Eight weeks afterwards and shortly before death an apoplectic attack accompanied by right facial monoplegia and followed by transitory aphasia; afterwards very defective thick speech. There was a dark brown hæmorrhagic softening, about two inches antero-posteriorly by an inch and a quarter laterally, in the anterior end of the left superior frontal and the adjoining part of the upper ramus of the middle frontal convolution.

Case I.—The ninth patient suffered from auditory hallucinations, was dull and slow, but free from paralysis until four months after admission, when she had an attack of left hemiplegia. Ten months after the commencement of this attack she could feed herself and was able to use both hands fairly well, but she was feeble generally and could not walk without assistance. Four months later still, an attack of left brachio-crural monoplegia; the right pupil was larger than the left; the patient protruded her tongue when asked to do so but never spoke after the commencement of the attack, which proved fatal in a week. Left cerebral hemisphere: (1) Superficial yellow softening of an inch and a quarter of the anterior end of the first temporal convolution, and half an inch of the second temporal; (2) Softening of the walls of the intra-parietal fissure over the supra-marginal and angular gyri, and partly of the small annectant gyrus. Right cerebral hemisphere: (3) Middle frontal convolution softened in posterior third, except a small portion near the ascending frontal, and the softened part contained a fresh clot about half an inch in diameter; (4) Outer half of orbital convolutions softened superficially, except half an inch posteriorly; (5) First temporal slightly softened on under (parallel fissure) surface for about an inch anteriorly; (6) Third temporal, superficial patch of softening an inch in length anteriorly, and very small patch behind this. Arachnoid thick on both sides. Patient was aged 60.

Case J.—The tenth case was that of a male patient, aged 50, who suffered from epilepsy as well as dementia, and who, six weeks after admission had an attack of left hemiplegia, in which the tongue alone escaped; there was at the same time convulsive twitching of the muscles of the left arm and leg; the right pupil was larger than the left; cutaneous sensibility (touch and pain) abolished on the left side, including the face; knee-jerks lost, no ankle clonus on either side; hearing unaffected on either side; vision of left eye lost; dull, heavy facial expression; no disorder of speech; left fingers extended and twitching. A day later the cutaneous sensibility had slightly returned, the left arm and leg were limp and flaccid, and there were frequent and sudden vomiting and retention of urine, yet the patient said he was very well. Two days later the gastric and vesical symptoms had disappeared and there was right conjunctivitis; on the following day the patient was restless and noisy, and there were visual hallucinations. Eleven days from the commencement of the attack he could stand without support and move his left arm. A week after the date of the last note he became feeble and drowsy and had sixty fits in eleven hours, remaining unconscious all the time; the convulsions affected only the muscles of the right side, face, arm, and leg, and there was conjugate deviation of the eyes to the right; each seizure lasted two or three minutes. The lesions were: (1) Insular sclerosis of the cortex of both cerebral hemispheres, the resulting circumscribed hardening of the tissue being readily perceptible to the touch in many places; (2) A focus of yellow softening in the cortex of the posterior part of the right gyrus fornicatus, measuring an inch and a half antero-posteriorly by half an inch vertically. The left cerebral hemisphere, free from membranes, weighed 652 grammes, the right only 585; each cerebellar hemisphere 82 grammes

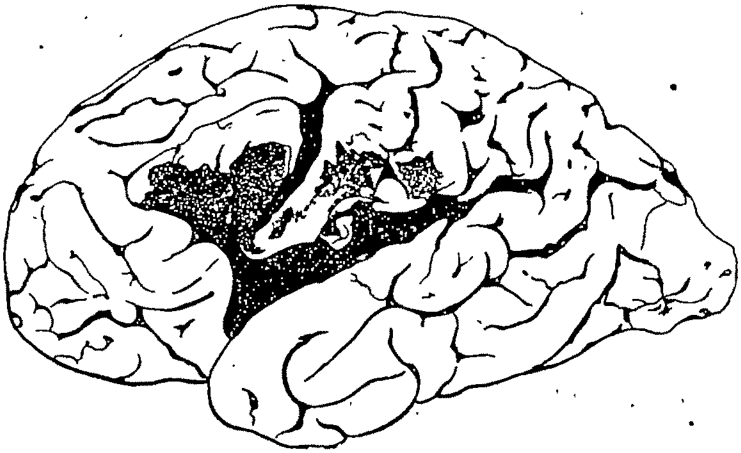


FIG. 2.—Case K.

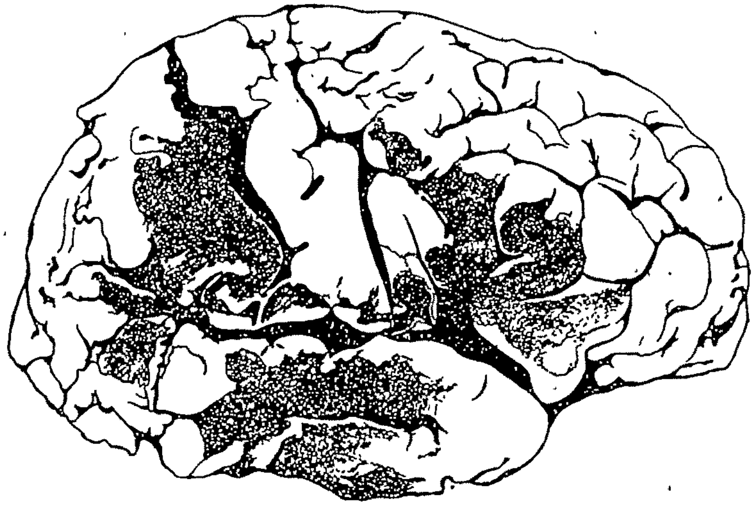


FIG 3.—Case K.

3 decigrammes, the pons and medulla 29 grammes 8 decigrammes; the whole brain, stripped of membranes, nearly 50½ ounces.

Case K.—The eleventh case was one of paralytic idiocy; there were brachio-crural monoplegia with contracture of both limbs, internal strabismus of left eye, total absence of speech and almost of intellect, epilepsy. There were cortical erosions on both sides of the cerebrum; the left hemisphere (fig. 2), much smaller than the right (fig. 3), was of such low specific gravity that it almost floated in weak bichromate solution; the left convolutions were less complicated, and their cortex paler than the right; the left central ganglia were smaller than the right; the right hemisphere of the cerebellum was the smaller; the left olivary body looked the larger owing to paucity of surrounding fibres, but on section the dentations were seen to be shrunken and irregular. The weights were: left cerebral hemisphere 212 grammes, right 342½, right cerebellar hemisphere 23, left 30·2, pons and medulla 20·3, all free from membranes, total 628 grammes (22⅛ ounces).

Case L.—The twelfth patient suffered from left brachio-crural monoplegia with brachial contracture and atrophy; she was 68 and had been paralysed 20 years; she could answer questions but not sustain a conversation; speech unaffected; right pupil slightly larger than left; slight deafness; no cutaneous anæsthesia; later, left crural contracture, and a fortnight before death, right crural. The only destructive cerebral lesion was a well-defined oval patch of yellow softening, measuring three-quarters of an inch antero-posteriorly, a quarter of an inch laterally, and one-eighth of an inch in depth in the cortex of the right supra-marginal gyrus; the frontal lobes were shrunken and some distance from the dura mater above them; the right hemisphere, though not weighed, felt appreciably lighter than the left; the cerebellum was soft and light coloured, especially the left lobe, and the roof of the fourth ventricle was soft and creamy. No other lesions.

Case M.—The thirteenth patient, aged 47, on admission, suffered from right facial and left brachio-crural monoplegia, the arm being most affected; motiveless weeping. Two months after admission he was quiet and understood what was said to him, but spoke hesitatingly and with difficulty, though to the purpose. A week before death he was seized with convulsions which occurred at frequent intervals until he died; they were confined to the right side and accompanied by unconsciousness and stertor: in the intervals he was conscious but quite unable to speak; the left arm partially recovered from the previous paralysis; the patient, when requested, tried to protrude his tongue but could not make it pass his lips: as far as he could put it forward, however, the tip tended to the right. Two days before death and the day before death he was conscious between the seizures but unable to move limbs, lips, or tongue. Right cerebral hemisphere: (1) Several old hæmorrhagic foci in the outer division of the lenticular nucleus and in the optic thalamus; (2) A large, brown focus with softening in the posterior limb of the internal capsule near its transition into the corona radiata. Left cerebral hemisphere contained: (3) a clot about 2 inches by 1½ in the corona radiata (also in the external capsule and lenticular nucleus), immediately outside of, and parallel with, the lateral ventricle in its anterior two-thirds; the clot caused the insula to appear dark, but did not actually rupture the cortex; the tissue round the clot was softened; there was fluid blood in both lateral ventricles.

Case N.—The fourteenth case was that of a paralytic idiot, aged 14, who was also hydrocephalic, porencephalic, and epileptic. He suffered from right brachio-crural monoplegia with contracture of both limbs and atrophy most marked in the arm; slight right internal strabismus; total absence of speech. He was about as intelligent as an average child a year old. In the status epilepticus preceding death the convulsions were not localised and there was conjugate deviation of the eyes to the left. There was an abnormally large quantity of serous fluid in the cranial cavity and the right cerebral hemisphere (fig. 4) was large with greatly dilated lateral ventricle, but otherwise normal; the left hemisphere (fig. 5) was very small and had the following convolutions and parts of convolutions and lobes missing, viz., the posterior half of the second frontal, the third frontal except its orbital portion, the inferior halves of the two ascending convolutions, the supra-marginal and angular gyri, the insula, most of the external surface of the occipital lobe. The centrum ovale under these parts was

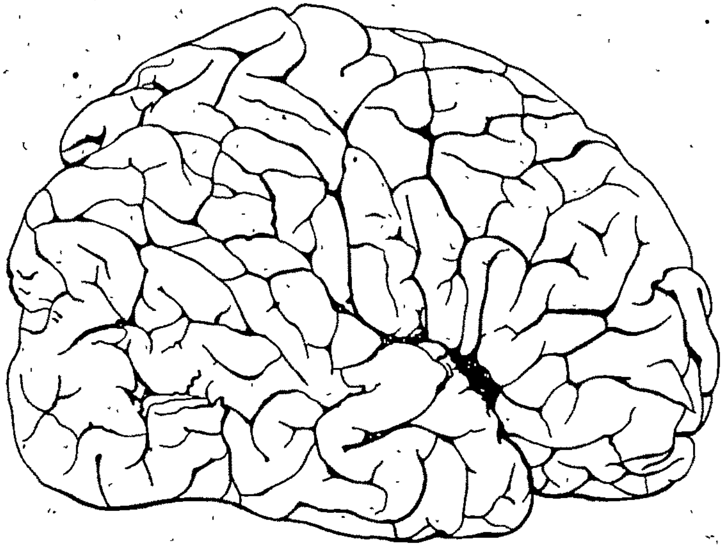


FIG. 4.—Case N.

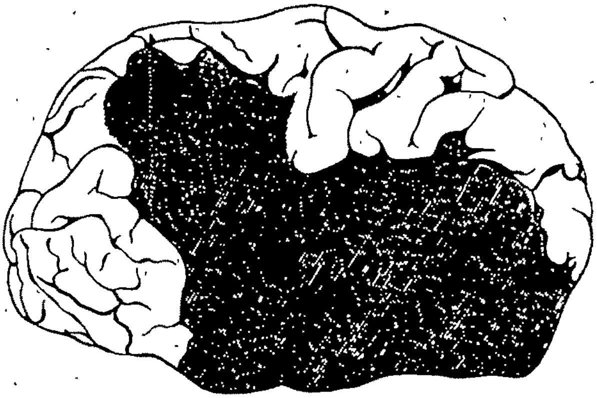


FIG. 5.—Case N.

represented by a thin lamina, translucent in places, and the site of the missing tissue was occupied by a sub-arachnoid cyst. The following parts were very much smaller than those of the opposite side, viz., the left central ganglia, peduncle, half of pons, half of medulla, pyramid; the right hemisphere of the cerebellum was much the smaller, the dentations of the left olive were small, shrunken, and irregular, and the right restiform body was apparently smaller than its fellow. The left cerebral hemisphere weighed 183·4 grammes, the right 523·2, the right cerebellar 48·6, the left 64·5, the pons and medulla 16, all free from membranes, total 835·7 (nearly 29½ ounces)..

Case O.—The fifteenth patient was also a paralytic idiot (aged 19); he suffered from right brachio-crural monoplegia and left crural with contracture of all the paralysed limbs; he could move his right fingers a little; the left fingers and hand were freely movable, also the left forearm, except that it could not be fully extended; the movements of the left shoulder were restricted. Mentally he was rather better than an ordinary year-old child, but could not articulate a syllable. Epilepsy, nystagmus; no sensory defects; emaciation of all the limbs. Atrophy or arrested development of the following convolutions and parts of convolutions, namely, lower two-thirds of left ascending parietal (which was very short), lower third of left ascending frontal except a quarter of an inch at the extreme end, left inferior frontal, an inch of the lower ramus of the left middle frontal, beginning a quarter of an inch from the posterior end, anterior half of left first temporal, anterior half of left second temporal, an inch of right ascending parietal commencing an inch from its upper end; left Sylvian fissure (owing to smallness of surrounding convolutions) gaping so as to expose nearly the whole of the insula; right occipital convolutions somewhat smaller than left; left central ganglia smaller than right; crus, pons, and pyramid less developed on left side. Weights: Left cerebral hemisphere 297·9 grammes, right 365·4, right cerebellar 68, left 69·8, pons and medulla 23·4, all stripped of membranes, total 824·5 grammes or a little over 29 ounces.

Case P.—The sixteenth was another case of paralytic idiocy with epilepsy; there was right brachio-crural monoplegia; the arm was principally affected and it alone was contracted; despite the weakness of the right leg the patient could walk a few steps; the right limbs were thinner and shorter, though not much, than the left; the patient could feel, see, taste, and hear, but not speak; his intelligence was not equal to that of an average child of one year. Left side of skull slightly thicker than right; left cerebral hemisphere shorter than right; both occipital lobes small and constricted looking; left temporo-sphenoidal lobe small and hard; left precuneus very small and narrow.

There were three cases of hemiparesis with general weakness, or rather of general weakness more pronounced on one side.

Case Q.—In one of these cases there was a history of a "stroke" eight months before admission, affecting "the legs principally," and followed by a second, third, and fourth confined to the right side. On admission there was general weakness, the right side being the weaker, the knee-jerks were exaggerated, there was marked right ankle clonus and the plantar reflex was absent on the left side; the pupils were very small but equal; the cutaneous sensibility was dull everywhere; there were motiveless weeping and laughter, visual hallucinations, and delusions of suspicion. A fortnight after admission there was coarse tremor of the right arm and leg, and the right arm was very feeble. Later, left facial paralysis, the nose being deflected to the right, right pupil the larger. Erysipelas, gangrene, œdema, bedsores. (1) Thickness of arachnoid and slight adherence over outer surface of both occipital lobes; (2) Triangular patch of softening with loss of substance of the right cuneus, the base, situated posteriorly, measured an inch and was an inch and a half from apex of focus; (3) Softening with loss of substance of rest of median surface of right occipital lobe; wide right calcarine fissure; (4) Small depression with softening on inferior surface, and another on outer border, of right lobe of cerebellum; (5) Partial sclerosis of right pyramid and left restiform body. The left cerebral hemisphere weighed 531·3 grammes, the right 518·4, the right cerebellar 64·8, the left 71·4, the pons and medulla 22·6, after removal of membranes.

Case R.—In the second of these cases, that of a female aged 38, all the limbs were weak, the left the weaker and very flaccid; the patient could not grasp with the left hand; slight drooping of left angle of mouth; left pupil the larger; knee-jerks absent, no clonus; general cutaneous anæsthesia; left foot swollen; epilepsy, the convulsions being severe and affecting the whole of the right side and the left upper arm. Motiveless weeping and laughter; capriciousness about food. Autopsy, 15 hours after death:—Pia congested; gyrus hippocampi on both sides soft and livid, the softening and discolouration extending into the gyrus uncinatus and cornu ammonis; similar softening and discolouration of tip of left temporal lobe; small portion of posterior part of each optic thalamus similarly softened and discoloured; cerebellum congested and dark. Stripped of membranes the right cerebrum weighed 478·2 grammes, the left 479·5, right cerebellum 71·4, left 68·7, pons and medulla 27·4.

Case S.—In the third case, the patient, a man of 66, was very feeble and tremulous generally and the right side was the weaker; cutaneous sensibility dull, especially on right side; tongue tremulous; vision very defective, but there was double cataract; left pupil larger than right; auditory hallucinations; memory very defective; said his wife or someone else drugged him and caused him to be weak. Eleven months after admission right side twitching, head deviated to right, complete loss of speech, temporary inability to move right arm; three epileptiform seizures. Two months later able to sit in a chair but legs very weak. (1) Patch of yellow softening, with loss of substance, an inch and a half by three-quarters, on the under surface of the left cerebral hemisphere affecting the hippocampal and the two occipito-temporal gyri; (2) Yellow softening of cortex of left cuneus; (3) Old hæmorrhagic focus with surrounding softening in posterior part of medullary substance of left frontal lobe near centre of vertical section (pedunculo-frontal); (4) Dark yellow softening of the right cerebellar hemisphere posteriorly, inferiorly and externally, and on lateral section a focus of ancient hæmorrhage an inch and a half long. Weights without membranes: Left cerebral hemisphere 526·8 grammes, right 567·6, right cerebellar 82·3, left 80·3, pons and medulla 23·3, total 1,280·3 grammes.

There were six cases of paraparesis or paraplegia with general weakness or rather of general weakness with the loss of motor power more marked in the legs than elsewhere.

Case T.—In the first of these the lower extremities were much attenuated, the gait was feeble, and the right arm and leg were weaker than the left; pruritus of lower part of thorax and upper part of abdomen; extreme mental weakness; motiveless weeping. A few days before death, slight left facial paralysis. (1) Large, yellow-edged excavation on the basal surface of the right cerebral hemisphere (fig. 6), affecting the inferior temporal and the occipito-temporal convolutions; (2) Small lacuna one-sixteenth of an inch in diameter in the outer margin of the external division of the left lenticular nucleus near the middle from before backwards.

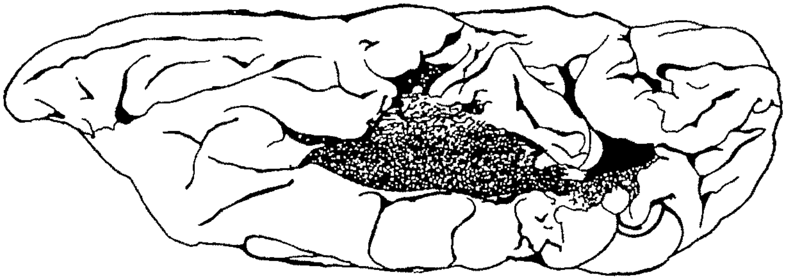


FIG. 6.—Case T.

Case U.—The second case was that of a female, aged 47, who, on admission, understood nothing said to her, was given to motiveless weeping, and suffered from general physical weakness, yet was occasionally violent. A month before death she became paraplegic and totally unable to speak. Four days before death there was left facial paralysis with slight left ptosis, and the right pupil was dilated. (1) Deep porencephalic cavity, measuring an inch and a half by an inch, involving the right supra-marginal, the angular, and the adjoining ends of the two upper temporal gyri; (2) Yellow softening of the right external occipital cortex with softening and lacunation of the white substance underneath; (3) Softening of cortex of left middle and inferior frontal convolutions with adhesion of pia arachnoid; (4) Softening, with arachnoidal adhesion of the upper (anterior) third of the left precuneus; (5) Focus of yellow softening, a quarter of an inch in diameter, in the anterior half of the left lenticular nucleus.

Case V.—In the third case the gait was feeble and awkward, and the cutaneous sensibility was dull on both sides; the knee-jerks were persistently absent, and there was no ankle clonus; the patient was restless, sleepless, excitable, and violent, but there were no convulsions during her three months' residence in the asylum, and no history (the patient was a married female, aged 60) of any prior to admission. Eight days before death the left pupil was larger than the right; the cutaneous sensibility was absent everywhere; both hands were feeble, though the patient could move her fingers; she said "yes" to every question; the left upper arm was a quarter of an inch less in circumference than the right, and the left thigh half an inch less than the right. (1) Tumour, an inch and a quarter in diameter, attached to the under surface of the dura mater, close to the right side of the falx cerebri, and pressing on the arachnoid over the upper ends of the right ascending convolutions, causing an indentation of the latter; (2) Arachnoid cyst, extending from the anterior third of the frontal convolutions to the parieto-occipital fissure on each side, for about 2½ inches from the median line, the membrane being very thick in places, especially near the falx and under the tumour. Weights of parts, free from membranes: Right cerebral hemisphere 405·6 grammes, left 401·7, each hemisphere of cerebellum 58·3, pons and medulla 19·4, total 943·3—a little over 33¼ ounces (the circumference of the head was 21½ inches).

Case W.—The fourth patient, aged 55, had, in addition to feebleness of lower extremities (not preventing walking), weakness of grasp, and slight left facial monoparesis; she suffered from epilepsy, emotional hyperæsthesia, and periodical attacks of excitement lasting several weeks; her vision was defective; strong erotic tendency. Two years and a quarter after admission a series of fits was followed by right facio-lingual monoplegia; the left knee-jerk was exaggerated and there was ankle clonus on both sides, more marked on the left; left leg the weaker; cutaneous sensibility retained. (1) Superficial yellow discolouration, varying in shade and unaccompanied by softening, of the following parts: posterior half of right superior frontal convolution, whole of right middle frontal, right central convolutions opposite base of middle frontal, whole extent of edges of right sulcus of Rolando, right supra-marginal gyrus, anterior two-thirds of right middle temporal at edges of small secondary furrows anterior third of right superior temporal; (2) Brownish-yellow discolouration, softening, atrophy, and depression of the right upper temporal for about an inch opposite the lower ends of the central convolutions; (3) Arachnoid adherent over the right occipital lobe; (4) Focus of softening an inch in diameter, affecting the adjacent portions of the left cuneus, precuneus, and two parietal lobules, destroying the cortex of the first annectant gyrus, and implicating the intraparietal and parieto-occipital fissures. The right cerebral hemisphere, free from membranes, weighed 550·8 grammes, the left 570·2, the cerebellum, pons, and medulla, 174·9, altogether, 1,295·9—45¾ ounces nearly.

Case X.—The fifth patient was aged 62, and had suffered for at least nine months prior to admission from extreme mental and general physical weakness, with paraplegia. On admission, the grasp was weak, the right pupil was the larger, and there were dysphagia and motiveless weeping. Two months later he had an attack, terminating fatally, in which he was conscious and made signs but could not speak, the mouth was drawn to the left side and the right angle drooped; swallowing was accomplished with great difficulty; the left pupil now became the larger. (1) Recent durhæmatoma over the parietal, temporal, and posterior two-thirds of the frontal lobes of both hemispheres, thickest over the left frontal lobe; (2) Superficial yellow softening of the right cuneus and the two occipito-temporal (fusiform and lingual) gyri. The cerebral arteries and the cardiac valves were very atheromatous, and the heart weighed 17½ ounces. The right cerebral hemisphere weighed 520·9 grammes, the left 546·9, each cerebellar hemisphere 71·4, the pons and medulla 19·4.

Case Y.—In the sixth case there were, in addition to the weakness of the lower extremities more marked on the right side, general feebleness, marked weakness of grasp, right facio-lingual monoplegia, very defective speech, and right facial twitching; the right pupil was the larger; the right knee-jerk was absent; there was some ankle clonus on both sides; vision very defective, enabling the patient to do no more than distinguish light from darkness; no deafness, either psychical, verbal, or ordinary. Two months later, right angle of mouth higher than left, but nose deflected to left and tongue protruded to right; in walking, patient leaned to right. Intense flushing of the face after taking warm beef tea. (1) Yellow softening of the right supramarginal gyrus, deepest at the margin of the Sylvian fissure where it affected the whole depth of the cortex, this focus extended well into the superior (posterior) extremity of the first temporal convolution; (2) Patch of yellow softening half an inch in diameter at the junction of the upper with the middle third of the left first temporal, affecting in its centre the whole depth of the cortex; (3) Atrophy of left lenticular nucleus with the middle division represented, on section, by a mere line dark in colour; (4) Asymmetry of cerebellum, the right hemisphere being the smaller; this hemisphere was yellow inferiorly; (5) Depression on the under surface of the right side of the middle lobe of the cerebellum. Weights without membranes: Right cerebral hemisphere 626 grammes, left 576·7, right cerebellar 64·8, left 72·5, pons and medulla 17·5, total 1,357·5, or 47⅞ ounces (circumference of head 23¾ inches).

Case Z.—This was a case of general weakness with left brachio-facio-lingual monoparesis which had commenced six months previously as left hemiplegia and developed ten months after admission into complete diplegia. The facial paralysis was rather right-sided on admission, but twenty days later was left-sided. On admission, the cutaneous sensibility was diminished on the right side, left pupil slightly the larger, watch not heard close to left, or an inch and a-half from right ear, left knee-jerk exaggerated, ankle clonus on the left side (the right ankle was stiff from injury and operation twenty years before), lingual and facial tremor, tremor of trunk muscles, lisping articulation, delusions of suspicion and jealousy, suicidal and homicidal tendencies, memory not extremely defective. History of alcoholism. After the second attack, three weeks before death, the cutaneous sensibility was slight on the left side of the face, and absent on the right as well as in the hands and arms, pin-pricks caused voluntary and reflex movements of legs, hearing absent on right side, slight on left, pupils dilated, the left the more so, inability to speak or to move lips, tongue, hands, or arms. (1) Softening of anterior half of right lenticular nucleus with lacuna a sixth of an inch in diameter in the anterior limb of the internal capsule; (2) Brown softening of the anterior half of the outer division of the left lenticular nucleus; (3) Recent clot a quarter of an inch in diameter in the upper part of the left external capsule, encroaching on the lenticular nucleus at the beginning of its posterior third; (4) Adhesion of pia-arachnoid to basal surfaces of cerebrum and cerebellum. Weights of parts, free from membranes except their inferior surfaces: Right cerebral hemisphere 570·2 grammes, left 583·2, right cerebellar 60·9, left 70·6, pons and medulla 25·2.

Case AA.—A case of chorea in a woman aged 69. The choreic movements were most marked in the arms, particularly the left. During a fatal attack of pneumonia the choreic movements diminished and there was sudden twitching of the head and neck at times; there were also slight right ptosis, right brachial monoplegia, and, four days before death, complete loss of speech. Several small lacunæ on both sides in the outer division of the lenticular nucleus, one on each side, situated inferiorly, one-sixteenth of an inch in diameter; on the left side many lacunæ in the external capsule, especially posteriorly; occipital arachnoid adherent, and many dark points in occipital medullary substance; medullary substance of cerebellum, grey in colour, with numerous small dark points. The left cerebral hemisphere weighed 460 grammes, the right 466·6, each hemisphere of the cerebellum 51·8, the pons and medulla 22. Cardiac mitral valve thick and opaque, and containing calcareous spicula. Knee-joints large, the internal condyles being unusually prominent.

There were three cases in which there were extensive destructive lesions without paralysis of limbs or face.

Case BB.—In one of these there was general weakness, but the patient was aged 75, alcoholic, and suffered from gastric cancer. His head was always bent down; motor speech was unaffected, but the patient rambled incoherently and self-contradictorily, and his memory both for recent and long past events was very defective. (1) Softening of the lower ramus of the right middle frontal immediately under the cortex and extending from the middle back to the frontal ascendant three-quarters of an inch in breadth vertically by quarter of an inch in depth; (2) Softening of the whole of the right inferior frontal (which was small), deepest in the centre; (3) Softening of the middle of the right insula effacing two or three of the digitations; (4) Arachnoid cyst from middle of frontal lobes to tips of occipital about a quarter of an inch deep at the deepest part. Right cerebrum 518·4 grammes, left 554·7; left cerebellum 64·8, right 68·7.

Case CC.—In another there was also general weakness with a tendency to hang the head constantly. The only other motor symptoms were those of unilateral laryngeal palsy, viz., lowness of pitch and hoarseness of voice; the patient answered questions in jerking monosyllables, and though generally listless and apathetic, he had explosive outbursts occasionally in which he talked thickly and very indistinctly so that only those accustomed to hear him could tell what he said. He knew common articles such as tobacco and pepper by sight but not by smell. There was either amblyopia of the left eye or left-sided hemianopsia. Large deep focus of ramollissement in the left cerebral hemisphere (fig. 7) affecting the middle third and part of the posterior third of the inferior ramus of the superior frontal convolution, the middle third and part of the posterior third of the second frontal, the posterior part of the inferior frontal near its junction with the frontal ascendant destroying a quarter of an inch of the cortex right across the convolution, a small portion of the middle of island of Reil. Left anterior cerebral artery much smaller than right. Arachnoid slightly adherent to occipital lobes. No other anomalies.

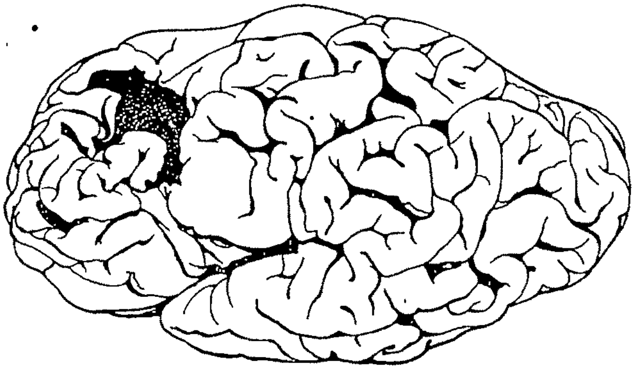


FIG. 7.—Case CC.

Case DD.—The third patient understood nothing said to her, but seemed to know those about her, and to hear so far as mere sound was concerned; latterly her vision became somewhat defective; she repeated constantly a few phrases such as "I lathers and shaves," "I'm a good maker here," &c.; she sometimes went through a pantomime of lathering and shaving; screamed when thwarted in any way; sang and danced in the daytime; restless and noisy at night. Yellow softening of the anterior half of the left temporo-sphenoidal cortex, that of the superior temporal alone escaping; right temporo-sphenoidal cortex in a similar condition for an inch anteriorly, the first convolution here, also, being intact; arachnoid cyst extending from posterior extremities of cerebral hemispheres to frontal ascendant on left side and parietal ascendant on right, attaining over the former convolution a depth of half an inch, over the latter of a quarter of an inch.

Summary.—In these thirty cases, given in very brief abstract, general physical and marked mental weakness with persistent slight paresis or transitory attacks of paralysis indicated, according to the combination of symptoms, lesions single or multiple of the occipital, temporal, supra-marginal, precuneate, or prefrontal regions, or of the cerebellum; whilst permanent paralysis with or without contracture or atrophy was the outward expression, or in some of the idiots perhaps the cause, of disease of the Rolandic area, or, though rarely, of the convolutions (including the precuneus and first temporal) immediately behind or below it, or of the central ganglia or internal capsule (disease in the ganglia and capsule being generally combined with extra-Rolandic, cortical or medullary lesions).

Except, perhaps, in the case (CC) of laryngeal palsy (not laryngoscopically determined) the exact situation of the lesion could not have been foretold by the greater or less degree of paralysis of any member or muscle; not only so, but the pathological appearances accompanying paralysis of a given limb were in some cases absolutely contradictory of those in others; there was complete hemiplegia with lesions confined to the upper end of the Rolandic region and its immediate vicinity in front (Case B), brachio-crural monoplegia with freedom from disease in that precise position (Case L), paralysis with contracture of one arm and both legs with atrophy of the lower two-thirds of the ascending parietal and part of the lower third of the ascending frontal on one side and of the middle third of the ascending parietal on the other (Case O), (the "explanation" that the so-called "centres" in the child differ from those in the adult, or something practically to that purpose, is anything but satisfactory, and, at all events, only the last of the three examples above was congenital or quasi-congenital). Whilst in some cases (L, P, &c.) of permanent paralysis there was no Rolandic destruction or atrophy, and in cortical facial paralysis (B, H, Q, T, U, X, Y, &c.) the seat of lesion varied widely, yet in no instance was there destruction or atrophy of the ascending convolutions without permanent paralysis.

When the pupil on the paralysed or paretic side was the smaller there was almost invariably ramollissement of the opposite cerebral hemisphere (Cases L, U, Q, R, X, S, Y, J), in one case of softening of the right hemisphere the left pupil was the smaller until, shortly before death, a durhæmatoma pressing more on the left hemisphere, caused a reversal of the relative size of the pupils (Case X). This partial exception (Case X) and another (also a case of durhæmatoma) not reported here, were the only ones to this rule. The pupil on the weak side being the larger (Cases H, V, D, I, F, G) seemed to denote destructive lesion or atrophy of the opposite central ganglia or irritative lesion (hæmorrhage, abscess, tumour, &c.) of the opposite cerebral cortex. In Cases U and Y there was lenticular lesion opposite to, and cortical on the same side as the larger pupil. In Case F the larger pupil was at first opposite to the older, then to the newer striate lesion.

Late pseudo-bulbar symptoms in three cases (M, X, and Z) pointed to severe bilateral lesions including hæmorrhage. In two of these, M and Z, the lenticular nuclei were diseased.

Whilst exaggerated knee-jerk and the presence of ankle clonus denoted, as a rule, either cortical or ganglionic destruction, the absence of these symptoms or of the normal knee-jerk, was explained by the presence of a pressure lesion, *e.g.*, tumour (V), hæmorrhage (in one case, G, these symptoms, previously present owing to central ganglionic destruction, disappeared on the supervention of internal capsular clot), nodules, as in the unusual case, J, of multiple sclerosis, in which the disease was almost altogether confined to the cerebral cortex, of hippocampal inflammatory softening (Case R), or of cerebellar atrophy, Case Y (knee-jerk absent, clonus present).

Tremor and twitching were generally the expression of disease (lacunæ, &c.) of the lenticular nucleus (as in case of chorea, AA), or of abscesses, tumours, hæmorrhages, or nodules (in the case of multiple sclerosis there was twitching but no characteristic tremor) in the cerebral medulla or cortex, or of lesions of the cerebellum. Unilateral convulsions denoted irritation in or near the Rolandic cortex or pressure lesions of the medulla of the temporal or parietal lobe (in cases of mine not here reported, unilateral convulsions seemed to be caused by glioma or other irritative lesion of the temporal).

Cutaneous anæsthesia (Cases R, S, J, Q, Z, F, G, V, A) appeared to be the outward expression of disease of the limbic or falciform lobe, the nucleus caudatus, the posterior limb of the internal capsule, or of the Rolandic area. Case G tends to show that the capsular fibres transmitting sensation from the spinal foot centre to the cortex are anterior to those for the other parts of the lower extremity. Thoracico-abdominal pruritus in Case T appeared to be caused by destructive lesion of the inferior occipito-temporal cortex.

Auditory symptoms mostly originated in disease of the temporal lobe or its vicinity, but not necessarily of the posterior end of the first temporal gyrus, in fact, in one case, this part of the lobe was partly destroyed on both sides without deafness, either psychical or physical resulting (Y). Visual symptoms occurred with extensive disease anywhere behind the sulcus of Rolando. The defective vision, left amblyopia or left-sided hemianopsia, noticed in one case (CC) of (left) pre-frontal destructive lesion may have been caused by impaired nutrition of the left optic nerve The anosmia in the same case possibly arose from a similar starvation of the left olfactory nerve combined with inability to sniff with the right nostril. But such "explanations" are liable to be fallacious, as many of those, for instance, given before the differentiation of motor from sensory aphasia undoubtedly were.

A small destructive lesion, especially of the cerebral cortex, caused, after a time, a diminution in the weight of the hemisphere in which it was situated out of all proportion to the mere local loss of substance.

The only regions constantly affected (either softened or atrophied), when there was crossed atrophy of the cerebellum, were the prefrontal and lenticular; it was not always present when there was paralysis, and it occurred in one case (BB) where there was no unilateral weakness;

in at least two cases (K and N) it was accompanied by atrophy of the olive on the side of the cerebral lesion. In one case (Q) of right median occipital and right cerebellar ramollissement the right pyramid and the left restiform body were partially sclerosed. In Case N, with diseased left cerebrum and atrophied right cerebellum the left peduncle, pons, medulla, and pyramid were much the smaller, whilst the right restiform body was apparently somewhat smaller than its fellow. In Cases K and O, also, the crus, pons, and pyramid were markedly less developed on the left than on the right side.

In Case F, most of the left pyramidal fibres and some of the right were degenerated. But this degeneration may not have been wholly caused by the left-sided destructive lesions, as the right nucleus caudatus was also slightly diseased.

ON SYPHILIS OF THE NERVOUS SYSTEM.

[*With a Critical Digest.*]

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INTRODUCTORY GENERAL REMARKS.

NOT only as regards syphilitic affections of the nervous system, but also other constitutional syphilis, we may notice the tendency to improvement or sometimes to cure under appropriate treatment, and the natural tendency of syphilis to remission, but also to relapse. It makes a truce rather than a peace. Similarly may be taken what we may call the fickle uncertainty and variability as concerns its degree of amenability to treatment; the unusual dissociations wrought by it in symptoms and lesions representing more formal classical types of disease; and, on the other hand, the unwonted associations of different symptom-groups, and pathological changes; the selective tendency, eventuating in a strange picking out of this or that part, not systematically, but as if at random, like the incidence of successive assaults delivered, now upon this outwork, and, anon, upon that rampart, of the citadel of life.

And, long ago, Dr. Buzzard and others insisted upon the important indications of syphilis afforded by the multiple character of syphilitic nervous affections, and by the co-existence of two or more grave lesions of the nervous system, not necessarily connected; and, similarly, Dr. Hughlings Jackson held that a random association or a random succession of nervous symptoms is very strong warrant for the diagnosis of syphilitic disease of the nervous system. Dr. Gowers also indicated that true syphilitic lesions mostly conform to two conditions, one of time, the other of place; as to the former, the most characteristic symptoms of true recognised specific lesions being, as a rule, neither very sudden nor very slow and chronic; as to the latter—or place—the specific processes being outside the nerve elements, with no special relation to the nerve functions, having a random distribution of their effects, and as a rule not selecting for attack and isolating, from amongst others, structures which possess a common function; although it is more than possible that a mere post-syphilitic degeneration may occur in these.

Nevertheless, it is probable, we think, that some directly-produced, if not in one sense "specific," processes are by no means "outside the nerve elements."

As to the frequency of brain syphilis, about 12 per cent. according to Dr. J. V. Hjelmmann,[1] about 21 per cent. according to Fournier, with tertiary syphilitic diseases show brain symptoms of syphilitic origin. And the former states that, of those infected with lues, from 1½ to 2½ per cent. get brain syphilis, and this, although general paralysis and tabes are expressly excluded from the computation. More cases of brain syphilis occur in the first year of the syphilitic "diathesis;" and in each subsequent year the number making appearance gradually dwindles. Age accelerates the onset of brain syphilis: the older the patient the shorter the average interval between infection and it. Thorough early mercurial treatment prevents much brain syphilis that would otherwise occur. Mental strain or excitement, cranial injury, sexual or alcoholic excess; are the most frequent excitants to a *cerebral* localisation of syphilis; in this, playing a part even more important than their frequent one of bringing on the sudden outbreak of an already existing, but slumbering, brain affection. Similarly, a general nervousness or nervous constitution may assist in determining the cerebral localisation of syphilis.

Of cases of brain syphilis, perhaps a quarter recover, another fourth improve decidedly; at least half die, or only survive with grave disease. About half of the deaths occur within two years of the appearance of brain symptoms. The prognosis is worse after the age of about 40 than before it (*cet. par.*); the interval between infection and onset of brain syphilis does not in itself affect the prognosis, unless it is more than ten years, a delay which seems to be unfavourable in prognosis (Hjelmmann). The most favourable of the conditions or forms of brain syphilis for recovery are brain irritation, epilepsy, and paresis of cranial (basal) nerves; limb paralyses are less so; and least so are the severe diffuse symptoms. Early energetic specific treatment is the most potent favourable element in the prognosis.

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[1] "Om hjärnsyfilis, dess frekvens, kronologi, etiologi och prognos." Helsingfors, 1892.

From an important point of view, the morbid states issuing from syphilis are divisible into the "*specific*" and the "*para-syphilitic*" groups.

The *specific* arise from the specific action, processes, and lesions of syphilis.

The *para-syphilitic* have origin in syphilis, are products of its action, under its influence, and without it, in all probability, would not be present; but although thus proceeding originally from syphilis are not of syphilitic (specific) nature.

As para-syphilitic, Prof. Fournier classes:—

For acquired syphilis: pigmentary syphilide; acute hystero-neurasthenia of the secondary stage, and later neurasthenic symptoms; hystero-syphilis; tabes; general paralysis; a special form of epilepsy; and, of muscular atrophy.

For hereditary syphilis: many general or partial dystrophies and organic malformations (*e.g.*, dental); and physical or mental developmental defects, infantile types; cachexia; rickets; hydrocephalus; some cases of simple meningitis in early life; possibly some cases of true epilepsy; certainly juvenile tabes and juvenile general paralysis.

The syphilitic affections of the nervous system might be systematically dealt with in several ways. We might arrange them on their pathological bases. For this the time does not seem to be fully ripe. A division into acquired and hereditary has the inconvenience of overlapping, and therefore of necessary repetition on many points. Or, systematically, one might deal with syphilitic nervous affections by dividing them according as they are outcomes of the secondary or of the tertiary stages of syphilis; but at the expense of some overlapping, repetition, and indefiniteness in the accurate placing of nervous symptoms in many cases; for the secondary and tertiary stages and their manifestations are not always distinct and separate; not only may they mingle but they may be separated by an interval or stage of "latency or of relapses" (Mr. Hutchinson); during which may be silent irregular workings, or creeping approaches, of syphilitic nervous conditions. And another objection to this plan of division is that syphilitic affections are apt to occur in persons in whom the outward manifestations of syphilis (*e.g.*, in skin, mucous orifice, or bone) have been short, slight, or absent.[1] On the other hand, and complemental to these in signification, is a group of cases, some examples of which I published[2]—namely, cases of visceral and other (bony, &c.) syphilitic lesions in insane patients, without cerebral syphilitic lesion; in some of which syphilis may, in others may not, be an ætiological factor of mental disorder by way of toxæmia, or of syphilitic cachexia and anæmia, or may have a subsequent modifying influence on the character of the mental symptoms by way of those, or of pain.

A convenient division of the material in hand is into six Parts.

Of these, *the first* consists of cases presenting a complex clinical study; part of which are cases of some severity, more or less threatening life; possessing multiple lesions, usually both cerebral and spinal in distribution, though in some intracranial only; and representing a widespread or manifold, yet more or less irregular, attack on the domain of the nervous system.

The third concerns syphilitic spinal cord, &c., disease, particularly that occurring alone or as the predominant feature of syphilitic nervous disease; especial consideration being given to syphilitic spastic spinal paralysis; and to taboid symptoms.

With this fuller statement as to Parts I. and III., the whole of the proposed series of them may now be briefly stated as follows:—

I.—Some forms of extensive, severe or multiple, mainly acquired, syphilis of nervous system, and chiefly multiple cerebro-spinal.

II.—Some aspects of hereditary or congenital syphilis.

III.—Syphilitic spinal cord disease, especially that evinced by spastic paralysis; or by tabetiform manifestations.

IV.—The relations between syphilis and tabes.

V.—Syphilitic epilepsy (so-called).

VI.—Syphilitic neuralgia (so-called).

Most concisely stated, these forms of syphilis of nervous system are the:—

I.—Cerebro-spinal.

II.—Hereditary.

III.—Spinals.

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[1] Buzzard, Broadbent. Also present writer's experience, *British and Foreign Med. Chir. Review*, July, 1876, p. 161-2. Similarly, Hjelmmann recently finds the preceding syphilis slight or moderate in intensity, as a rule; and Hougberg declares it relatively mild.

[2] *Journal of Mental Science*, January, 1884, p. 492. Read at annual meeting of B.M.A., August, 1883.

IV.—Tabetic? (question of).
V.—Convulsive.
VI.—Neuralgic.

These are variously associated in some cases. They will be taken in order. But there is not space for an exhaustive inclusive dealing with any part of the subject. For convenience, these Parts or Divisions are sub-divided into Sections, which are indicated by capital letters.

PART I.

SOME FORMS OF EXTENSIVE OR MULTIPLE SYPHILITIC DISEASE OF NERVOUS SYSTEM.

As already stated, these are chiefly multiple cerebro-spinal, and, although mainly acquired, are sometimes hereditary and conveniently taken here.

A.—*General Paralysis of the Insane.*

Elsewhere, I[1] stated that:—

"There are several possible ways in which syphilis may conduce to general paralysis.

"(1) The syphilitic diathesis may weaken the resistive power of the organism at large, or brain particularly, to causes of general paralysis, and thus dispose to that affection.

"(2) Other influences, acting on the brain and nervous system of a syphilised person, may rouse the latent tendency to syphilitic disease, in the parts then placed under strain.

"(3) Syphilis may, perhaps, directly produce the inflammatory changes of brain" (*i.e.*, in general paralysis).

According to the *third* of these relationships between syphilis and general paralysis, the latter would be accepted as a true syphilitic lesion resulting from the direct effect of syphilis in producing a pathological change in the brain. According to the *first* of them, syphilis would be taken as merely predisponent, weakening the organism at large, the brain- and cord-meninges particularly, the walls and vaso-motor state of their blood vessels especially; thus lessening their resistive power to the factors actively concerned in the production of general paralysis, and permitting of easier proliferation in, or exudation through, the vascular walls, and promoting a block and stasis in the lymph-canalicular system of the brain. In other cases, the similarly preparing and predisposing factors may be, not syphilis, but alcohol, lead, &c., and other forms of toxæmia, whether proceeding from perverted metabolism, certain noxious foods, or the toxines of other pathogenic microbic origin.

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[1] "General Paralysis of the Insane," second edition, 1886, p. 274.

Statistics collected by Rieger[1] show that, comparing 1,000 cases of general paralysis with 1,000 other cases, ten times more of the former than of the latter were believed to be syphilitic; or as 399 and 39, respectively, per 1,000. Another observer found a syphilitic history seven to ten times as frequently in general paralytics as in other insane; another alleges nearly 18 times as frequently.

Most differing, and even irreconcilable, have been the percentages in which general paralytics have been alleged to have had syphilis. Among the recent, are two very extreme and opposing statements; the one making only a fraction over one per cent. of general paralytics to be subjects of syphilis; the other making 93 per cent. to be so, and coupled with the assertion that the relation between the syphilis and the general paralysis is one of cause and effect. Perhaps we need not take either extreme statement too seriously.

In general paralytics, Kundt[2] found syphilitic infection certain in 8·2 per cent. and probable in 4·3 per cent. more; Hougberg[3] stated it at 74 per cent.; Tchige,[4] at 74 per cent., for certain, and probable in a few more. Recently also Régis[5] states the proportion of general paralytics with antecedent syphilis at 85 per cent. Christiani[6] puts it at nearly 63 per cent.; but in all but 10 per cent., other causes of general paralysis were coexistent. On this basis, it seems a little strange that "no prostitutes were affected"; yet the same incongruous result also crops up in the returns of Siemerling, and of A. Westphal.[7] And here may be mentioned the case of a woman who infected five youths with lues, three of whom died of general paralysis and two of brain syphilis. She got well, married, and has two children alleged to be healthy (Morel-Levallée).

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[1] "Schmidt's Jahrbucher," 1886, Bd. i, p. 88.

[2] *Allg. Zeitschrift für Psych.*, Bd. L.

[3] *Ibid.*

[4] Congress of Russian Physicians, December 28, 1893.

[5] *Archives Cliniques de Bordeaux.*

[6] *Rivista Sperimentale di Frenatria*, xix., Fasc. ii.

[7] *Charité Annalen*, 1898.

Kowalewsky[1] found brain lues in some cases without syphilitic history; and, on the contrary, that in some cases with luetic history other agencies were the effective ones.

Dr. H. M. Bannister[2] states that 72 per cent. of certain cases of general paralysis were undoubtedly syphilitic, and 17 per cent. more, probably so.

Dr. F. Peterson's[3] statistics (and not his only are constructed in like manner) make about 60 per cent. of the general paralytics admitted to a public asylum to be syphilitic : a conclusion arrived at by finding that in 19 per cent. "syphilis was definitely ascertained to have preceded the" general paralysis; and then by assuming that syphilis had been present in half of the remaining 81 per cent., or vast majority, of the patients; namely, those in whom syphilis was either absent or unascertained; and by adding that half, or over 40 per cent., to the 19 per cent. in whom syphilis *was* ascertained. Thus, he makes up a total of about 60 per cent., taken to be syphilitic, of which total, therefore, the major fraction of more than two-thirds represents, not ascertained facts, but an arbitrary assumption.

On the other hand, he has observed the practically total absence of general paralysis among the native Egyptians, in whom syphilis is a wide-spread disease, but in whom, owing to the interdiction by the Koran, alcoholism is seldom or never observed. Among the Arabs also, Pascalis makes the progress of general paralysis to be parallel with that of alcoholism.

Here we may refer to the old observation of the comparative rarity of general paralysis in Ireland and in the north of Scotland. Also, to the experience of Lewin, who in 20,000 cases of syphilis observed one per cent. become insane, but not a single general paralytic.

The first of the views mentioned at the beginning of this section, namely, that syphilis is only a predisposing cause of general paralysis, is a wide-spread disorder, weakening the constitution and vitiating the blood in many of its subjects, and thus preparing the system for the direct operation of the final ætiological factors of general paralysis, is the one adopted by Peterson. Moreover, in his view, not only is general paralysis not a form of specific disease, nor a late syphilitic manifestation; it is not even "a form of degeneration depending upon the syphilitic poison for its origin."

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[1] *Allg. Zeitschrift für Psychiatrie*, Bd. L.

[2] *Journal of Nervous and Mental Diseases*, 1891.

[3] *Medical Record*, New York, December 9, 1893, p. 741.

The percentages mentioned in the text are arranged below. They concern the proportion in which general paralytics were stated to have a history or indications of antecedent syphilis. Merely "probable" examples of this are added separately, in some of the statistics. Other statistics are summarised below, for comparison. Remarks on certain statistical methods used follow the table.

Jespersen ...	72·4 per cent.; and in 5 per cent. more, very probable.
Pontoppidan ...	33·3 per cent.; and in 5·3 per cent. more, probable.
Mendel	75·0 per cent.
Rohmell... ...	76·0 ,, ,,
Kundt	8·2 ,, ,, ; and in 4·3 per cent. more, probable.
Rieger	40·0 per cent. (collected statistics).
Œbeke	53.0 ,, ,,
Régis and Bonnet	85·0 ,, ,,
Bannister ...	72·0 ,, ,, ; and in 17·0 per cent. more, probable.
Peterson ...	45·0 per cent. at one institution.
,, ...	19·0 ,, ,, at another institution; and (?) per cent. more, probable.
Christiani ..	62·8 per cent.; with other causes in all but 10 per cent.
Hougberg ...	74·0 per cent.; and in some more, probable.
Tchige	74·0 per cent.; and in a few more, probable.
Savage	70·0 per cent.
Ziehen	33·0 ,, ,,

Average of 16 above=55·8 per cent. Most of the highest estimates are included (alleged "probable" cases not included).

Average of 16 others collected by the writer=45·6 per cent. Including many of the higher estimates.

Total average of 32 sets of statistics=50·7 per cent.

It is to be noted that these *averages* are only averages *of the percentages* stated by different observers; the actual number of *cases* to which some of them relate not being at hand.

FEMALE CASES ONLY.

Obersteiner ...	$1\frac{1}{8}$ per cent.
Jung	$3\frac{1}{8}$,, ,,
A. Westphal ...	7·4 ,, ,, ; and probable in 32·4 per cent.
Reinhard ...	28·7 ,, ,,
Jacobson ...	50·0 ,, ,, ; and probable in (?) per cent., more.
Jacobson ...	7·4 ,, ,, (collected statistics); uncertain in ·6 per cent. more.

As to the space of time between syphilitic infection and the onset of general paralysis, Hougberg places it mostly between five and nineteen years (both sexes); A. Westphal at one and a half to twenty-three years (women only). In most of my cases from five to twelve years; but in some of them a very much shorter time, intervened. Filliatier reports an interval usually of from ten to thirty years; but one at eight years; one at five; and one at one to one and a half year.

With regard to the above percentages in which syphilis or a syphilitic history is assigned to general paralytics, there are several points to mention; some of strained interpretation, and some of fallacy.

Some of the results have been arrived at by excluding all cases in which the history is not clear either way. This would strike out a great number of those not syphilitic, and thus unduly exaggerate the syphilitic element. In how few cases can one get a clear history of the *absence* of preceding syphilis in a more or less demented and facile general paralytic!

Then as to most of those stated to be syphilitic, there was merely a history of syphilis, or that which was taken to be so; and it would be begging the question altogether if one assumed that a subsequently occurring general paralysis was necessarily of syphilitic origin.

Again, in a number of instances, the percentages finally stated, or quoted and requoted, as representing those who had had syphilis, in the experience of certain observers, are partly constituted of cases in which syphilis was only suspected, or for various reasons was deemed to be more or less probable; cases which, nevertheless, are lumped together with those in which syphilis was definitely ascertained. Even worse than that, is a plan adopted of adding, and merely on surmise, a large fraction of the cases in which nothing clear was known either way, as to syphilis, to the percentages in which syphilis was definitely ascertained, thus classing, *as syphilitic*, cases of which nothing relevant is known, except that no history or symptom of syphilis was recorded or observed.

In some statistics, another error has undoubtedly arisen by the inclusion of cases as being of precedent syphilis, in which the latter was subsequent only; for, as I[1] stated, "many incipient general paralytics expose themselves freely and thoughtlessly to syphilitic infection, or in consequence of the increased sexual appetite occasionally observed as an early symptom"; and several such cases I have seen. Ten cases, in which the syphilis was thus incurred *after* the patients had become general paralytics, were reported by Kiernan;[2] and Ripping and Snell[3] made known examples of the kind.

There are some indications that some have included under the head of supposed syphilis examples of any kind of venereal sores, discharges, and non-specific herpes or glandular swellings, or later rashes, &c. A genital scar is most often from soft non-infecting sore. Soft non-syphilitic sores assume induration under various conditions. These facts explain certain sources of error.

Finally, it is extraordinary on what slender grounds some cases of general paralysis, or of tabes, have been attributed to syphilis, or set down as being in some way of syphilitic origin.

A history of antecedent syphilis alone, is sometimes tacitly assumed to show syphilis to be at the root of a later-coming general paralysis; a fallacy which involves two others, namely, that the person is necessarily still syphilitic, and that syphilis is incurable.

I do not dispute, on the contrary I fully admit, the vast importance of syphilis in the ætiology of general paralysis; but it is necessary to deprecate the method of computing the frequency of its action made use of in some instances.

It is comparatively difficult to get at the truth with regard to syphilis in *female* patients, especially those of the higher classes; and in the statistics of individual observers on this point the number of female cases is too small to draw conclusions from, except those of a few; such as Obersteiner,[4] who in 340 female general paralytics found only four (or $1\frac{1}{5}$ per cent.) syphilitic;

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[1] "General Paralysis," second edition, p. 274.

[2] *Alienist and Neurologist*, January, 1893, p. 56.

[3] *Allgemeine Zeitschrift f. Psych.*, Bd. xxxviii.

[4] *Wiener Med. Wochenschrift*, No. 33, *et. seq.*, 1883.

Jung,[1] who in 95 found three (or 3⅛ per cent.) syphilitic; A Westphal,[2] who in 148 found syphilis certain in 7·4 per cent., and probable in 32·4 per cent.; and Reinhard,[3] who in 87 found a history of syphilis in twenty-five (or 28·7 per cent.). Here, also, are the statistics of D. E. Jacobson[4] who (after excluding some demented cases from whom no information could be got), in 100 female general paralytics, found syphilis acknowledged in 37 per cent., and most likely in 13 per cent. more. Much like some others in this matter, by straining the interpretation of the facts, he raises the syphilitic percentage to a problematical 65 per cent. Here, the statements of the patients themselves appear to have been the chief source of information. But surely, most public asylum general paralytics (as these were) are considerably demented; are facile and modifiable in statement, easily shifting their ground on suggestion, and therefore readily, although unconsciously and unintentionally, induced to admit a non-existent syphilitic history, to the eager questioner, pre-occupied by certain conceptions on the point.

Since the preceding was written, it appears, in the course of an interesting paper by Dr. Ashby Elkins[5] on the twenty-eight adult female general paralytics admitted into an asylum during a term of years, that "seven of the twenty-eight are known to have led immoral lives, several others had certainly had syphilis, and twelve had been habitually alcoholic." Most of them had been good-looking, dressy, and of bad and dissolute, rather than good, lives.

For some years, the theory of the syphilitic origin of general paralysis, set up by Esmarch and Jessen, became, in its extreme and exclusive, or almost exclusive sense,[6] a Scandinavian theory; Steenberg declaring that there was no general paralysis without syphilis; Kjellberg that general paralysis was a form of cerebral syphilis, and never occurring in an organism free from both acquired and congenital syphilis; and Jespersen and others holding somewhat similar views.

The most important recent convert towards or to this theory or doctrine is Prof. A. Fournier, inasmuch as he was the champion of an opposing doctrine. The former, indeed, has been called the "pure" or "uniform" doctrine of general paralysis, in contradistinction to an alleged "dual" doctrine of Fournier. This last name seems to be not happily chosen. Fournier's point was not that there are two forms of general paralysis, but that the similar syphilitic brain disease is *not* true general paralysis. Moreover, Fournier's former view has recently been strained, and he has erroneously been represented as regarding the coincidence of syphilis and general paralysis in any case as purely accidental; and as giving the name of "general paralysis of syphilitic origin" to the syphilitic brain disease he described.

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[1] *Allgemeine Zeitschrift für Psychiatrie*, Band xxxv.

[2] *Charité Annalen*, 1893.

[3] *Allgemeine Zeitschrift für Psych.* Bd. xli., 1885, p. 458.

[4] *Journal of Mental Science*, April, 1892, p. 184.

[5] *The Lancet* (London), June 28, 1894, p. 1,496.

[6] See the writer's "General Paralysis," p. 278, second edition.

For this reason, and on account of the importance and historic interest of the subject, I will re-state Fournier's former, but now partially abandoned, doctrine by quoting part of a review I[1] wrote on his book:[2]—

"With reference to the much-debated question of the relations between syphilis and general paralysis he makes mention of two absolutely distinct groups. The one consists of cases in which true general paralysis of the insane supervenes in syphilitic subjects, but not in any unusual or undue proportion. As between the two extreme views—on the one hand, that in cases such as these, the syphilis is a mere accidental coincidence, and counts for nothing etiologically; and, on the other, that in them syphilis is directly pathogenetic—he takes up an intermediate position, believing that syphilis may aid in the production of such disease, not by acting as a specific cause, but as a diathetic one, and thus exerting merely an ordinary disturbing influence on the organism."

"The other group is that termed by him 'pseudo-general paralysis of syphilitic origin' or ('specific pseudo-general paralysis'); as examples of which he limns cases in which the symptoms, both psychical and somatic, and the morbid anatomy, read as do those of cases of general paralysis.

"But they differ from general paralysis, *as to symptoms*:

"(*a*) In the absence of ambitious conceptions.

"(*b*) In the absence, or slightness, of the trembling of lips and tongue, which, when present, is unlike the fibrillary vermicular tremor, and the twitching or quivering of general paralysis.

"(*c*) In the presence of true paralyses and pareses; particularly of partial strictly local paralyses, and of hemiplegia; while, on the other hand, at one moment he denies the occurrence of true palsies in general paralysis, at the next admits the occurrence of hemiplegia therein, although with excessive rarity only. . . .

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[1] *Journal of Mental Science*, April, 1880, p. 98.

[2] "La Syphilis du Cerveau," Paris.

"(d) Further differences exist: in *the mode of onset*, which in cerebral syphilis is often by apoplectiform or sudden paralytic seizures; in general paralysis usually by intellectual or moral disorders; in *the evolution and course*, which in cerebral syphilis are far more irregular and variable, both as to the manifestations and their succession; in *the duration*, ill-defined in the syphilitic; in *the appearance and condition*, which in cerebral syphilis are often cachectic; and in *the prognosis*, which in the syphilitic cases is by no means always or altogether hopeless.

"They differ from general paralysis also *as to the lesions*:—

"(e) In the predominant lesion being meningeal, a hyperplastic meningitis—a meningeal sclerosis—while, relatively, the lesion of the grey cerebral cortex is much less. Whereas in general paralysis the lesion of the grey substance is predominant, that of the meninges subordinate. The cortical lesion, also, is more circumscribed and localised in cerebral syphilis than in general paralysis."

And, in conclusion, I thought that "Fournier has included under the term 'syphilitic pseudo-general paralysis' both certain cases of syphilitic disease, resembling general paralysis of the insane more or less, and certain cases of true general paralysis. Furthermore, he refers to one kind alone of syphilitic lesion as bringing forth a resemblance to true general paralysis, while the writer of this review would assign that *rôle* to several kinds of syphilitic lesion, in different cases."

The difference between some of the pathological changes of brain syphilis and general paralysis is supported by examples cited (by Peterson) of necropsies in which "gummatous and arterial syphilitic processes existed side by side with the pathological changes of general paralysis." In two cases, published some years ago by the present writer,[1] there were those syphilitic processes, with some changes, as in general paralysis, irregularly distributed.

It is convenient here to refer very briefly to a cognate subject, *i.e.*, whether syphilitic true general paralysis differs from otherwise-produced true general paralysis; or, again, whether cases of general paralysis with a distinct history of antecedent syphilis, but without distinctly specific, generally-so-recognised lesions of syphilis of nervous system, have, or not, distinctive characters separating them from other cases of general paralysis.

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[1] *British and Foreign Med. Chir. Review*, July, 1876. Cases 1 and 2.

I concluded that the cases of general paralysis corresponding to that description fell into two groups, one of which did not, the other did, present such distinctive characters. The conclusions are too long to quote here. Those interested in the subject will find them at pp. 225-226 of my work, "On General Paralysis," already cited. But on the same subject, it may be added that my general view was confirmed by Schüle: he, indeed, seems to speak of distinctive characters as if always present; but that is not my thesis.

Recently, Œbeke,[1] also, comparing a syphilitic with non-syphilitic history in general paralysis, believes that equality of pupils, affections of sympathetic nerve-supply of eye, bilateral pupillary reflex immobility (as compared with unilateral, which is *less* frequent in the syphilitic), ptosis, depressive or melancholic symptoms, and dementia, are relatively *more* frequent in the syphilitic than in the non-syphilitic; and that in the former, also, the other ætiological factors are chiefly heredity, fatigues and sexual excesses; but in the non-syphilitic, mental strain and emotional disturbances. On the contrary, Hougberg found no clinical or pathological difference between the two, nor did Mendel.

The discussion on this subject is so often mixed with that of tabes that we cannot deal with them quite separately. Thus, in favour of the syphilitic origin of tabes,[2] Sachs speaks of the frequent development of general paresis after tabes, and of tabic symptoms in the course of general paresis, the latter also being much more apt to occur in syphilitic subjects than in others. But it cannot be admitted that that former development is *frequent*; *i.e.*, if we speak of true tabes and not of mere ataxic or tabetiform symptoms in general paralysis. (Four cases of general paralysis in the course of tabes are reported recently by Ph. Zenner;[3] exalted illusions and hypochondria in some of them. Of course, quite a number of such cases are on record altogether.) Nor can it be admitted that "cases of tabes, and for that matter of general paralysis as well, occurring in women and in very young subjects are invariably preceded by syphilis, which may be acquired or hereditary." This is frequent but not invariable. Another argument used is that cases of syphilis may resemble general paralysis, and, on the other hand, tabes.

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[1] *Allg. Leitschrift für Psych.*, 1. Band., p. 169. Also other differences not specified above.

[2] *New York Medical Journal*, January 4, 1894.

[3] *Cincinnati Lancet*, Clinic, May 18, 1893.

In the two diseases there does not, however, seem to be a very similar percentage of a history of syphilis, assigned by all observers, although it is by some. Thus Paterson, among certain patients, found 71 per cent. of tabetics with syphilitic history, and only 45 per cent. of general paralytics; the argument for a syphilitic ætiology of general paralysis, on the ground of a close relationship between it and tabes, he rightly holds not to be supported by those statistics.

Yet an argument in favour of the close connection between syphilis, tabes, and general paralysis, has been drawn from the parallel racial differences, observed in relation to all three, on comparing Russians with Russian Jews, both suffering from nervous diseases. Thus in 260 such Russian Jews, and 123 Russians, an indubitable history of syphilis is stated to have been found in 3 07 per cent. of the former, and in 16·2 per cent. of the latter (or over five times more per cent. of Russians than of Jews) —tabes in ·7 per cent. of the former, and in 3·25 per cent. of the latter (or nearly five times more per cent. of Russians than of Jews)—general paralysis in ·7 per cent. of the former, and in 4·8 per cent. of the latter (or about seven times per cent. more frequent in Russians than in Jews). By a clerical error, this is put as *five* times in the original.

But the combined statistics (including these) of Minor,[1] Kojewnikoff and Korsakoff show a closer approximation in the ratio between the percentages (of the total number suffering from nervous diseases), in which these two races are affected with tabes and with general paralysis, namely:—

With tabes ·86 per cent. (8½ per 1,000) of the Russian Jews.

With general paralysis ·86 per cent (8½ per 1,000) of the Russian Jews.

With tabes 2·9 per cent. (29 per 1,000) of the Russians.

With general paralysis 2·6 per cent (26 per 1,000) of the Russians.

The comparative rarity of general paralysis, of tabes, and of syphilis in Russian Jews, and the frequency of nervous affections generally, and therefore of the hereditary nervous element, in them, is taken to tell against the importance of the hereditary factor in general paralysis. Most of the general paralytic

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[1] *Archives de Neurologie*, tome xvii.

Russian Jews and especially the females, are stated to have had syphilis.

In relation to the above, it may be added that Strümpell looks upon tabes as the same morbid process as general paralysis, but possessing a different site, and that Jendrassik took much the same view; as also Fournier, recently. In tabes, Jendrassik found a lesion of brain cortex, like one found in general paralysis, but differing in site; and Nageotte,[1] in one out of three brains of ataxic patients without mental disorder, found, in the gyrus rectus and convexity of frontal lobe, cortical interstitial encephalitis as in general paralysis (cellular infiltration of vessel walls, neuroglia-cell proliferation, disappearance of tangential medullated nerve fibres).

According to this, general paralysis would be the cerebral, and tabes the spinal, form of what is thereby taken to be fundamentally one and the same pathological condition or disease; often originated by steady poisoning of the system following infection.

The rarity of general paralysis in gentlewomen may relate partly to the rarity of syphilis in them. The usually accepted comparative frequency of both of them in women who are of loose or promiscuous sexual habits, or who are the wives of sexually dissipated men, may have a closer connection than is at first apparent. Nevertheless, some instances of a striking absence of prostitutes from certain statistics of general paralysis, and therefore telling the other way, have been mentioned on a preceding page.

We next take up more distinctly the *third* of the possible relationships of syphilis to general paralysis, mentioned at the beginning of this section ("A"), namely, syphilis directly causing general paralysis, or a form of it, *i.e.*, lesions of true general paralysis considered to be specific syphilitic lesions.

Of forty-six cases of syphilis with insanity, many of them with gross syphilitic lesion of nervous system, published by the writer of this[2] up to some years ago, six cases were clearly general paralytic, and five more cases at least closely resembled general paralysis, the latter being such as are often (although not very happily) termed "pseudo-general paralysis."

Prof. F. Raymond[3] considers syphilitic general paralysis to be a chronic interstitial inflammation, having its point of departure in the capillaries of the brain, particularly of the cortex cerebri. The lesion, he terms diffuse embryonic meningo-encephalitis, or, otherwise, diffuse vascular meningo-encephalitis; and so terms it owing to the infiltration of capillary lymphatic sheath, as well as of lymphatic sheath and outer wall of larger arteries, and of musculo-connective tunic of veins, with embryonic cells possessing rounded nuclei (and not having nuclei of neuroglia cells). Thus, in the capillaries, is infiltration of adventitial sheath by embryonic cells with rounded nuclei; in the arteries, diffuse or nodular periarteritis, and ordinary secondary obliterative endarteritis (such as may follow periarteritis of any kind, and is a consecutive sclerotic lesion, anatomically that described by Heubner); in the veins, diffuse or nodular infiltration by similar cells in the musculo-connective tunic.

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[1] *C. R Soc. de Biol.*, January 28, 1893.

[2] Chiefly in *Brit. and For. Med. Chir. Rev.*, 1876 and 1877, and *Journ. Mental Science*, 1879 and 1880 With a few exceptions the cases observed of later years still await analysis and publication.

[3] *Archives de Neurologie*, Jan., 1894, t. xxvii.

In the cortex, also, is early disappearance of some tangential fibres.

Meningitis, also, is revealed by similar changes in capillaries and other vessels, sometimes with miliary gummata, *i.e.*, when the nodular character of the infiltration is unusually pronounced at points. The large blood vessels at the base of the brain, and their branches, are likewise often affected.

In the spinal cord, embryonic meningo-myelitis often coexists.

Wherever, in brain, cord, or meninges, the cell-infiltration forms accumulations—that is, nodular change in the vessel-wall—there is the first stage of a miliary gumma, which tends to become caseous, and thus a fully-formed gumma.

Thus there are, in the spinal cord, two syphilitic inflammations, the gummy meningo-myelitis and the diffuse embryonic meningo-myelitis. (But see also Part III.—W. J. M.) These meningo-myelitides may end in a sclerous (cicatricial), or in a sclero-gummatous change; or lead to foci of softening by the occurrence of thrombosis.

The two processes, above referred to, in reality only differ from each other by intensity and localisation; in the one case the embryonic elements only develop at certain points, but with an extreme abundance; in the other, the same elements proliferate much less actively, but over a far greater extent. The proof that there are not fundamental differences between them is that these two processes often combine to form a myelitis at once gummatous and diffuse. The diffuse is the form termed diffuse embryonic meningo-myelitis by MM. Gilbert and Lion.

But in the brain, while gummatous meningo-encephalitis is recognised by all [most ?] as syphilitic, the diffuse embryonic meningo-encephalitis has not yet received general recognition as a syphilitic lesion. Raymond urges strongly on behalf of its syphilitic nature, and that, in at least "syphilitic general paralysis," this diffuse embryonic meningo-encephalitis is to meningo-encephalic gumma, what embryonic meningo-myelitis is to gummatous meningo-myelitis; and that between true general paralysis and "syphilitic pseudo-general paralysis," are many intermedial conditions.

Other infections, probably, also occasion diffuse vascular changes, microscopically indistinguishable from these.

Case 1.—Female, aged 45; died in an epileptiform attack due to hæmorrhage at brain base, from rupture of a syphilitic vein. There were clear signs of early general paralysis, and tabetiform ones due to diffuse embryonic meningo-myelitis. Syphilitic lesions affected the large vessels of brain and cord. A diffuse vascular lesion affected the cerebral grey cortex, and in a less degree the subjacent white. It consisted of an accumulation of cells with rounded nuclei in the lymphatic sheath of the capillaries, and in the lymphatic sheath and wall of the larger vessels. These infiltrated embryonic cells belong to the mesodermal tissue; are fixed cells without nuclei of leucocytes; and are distributed in little irregular clusters or groups on the adventitia of capillaries (nuclei of endothelium and of muscular tunic not affected) Neurogliar changes and spider cells only slightly marked. Whereas the nerve cells were but little affected, the tangential fibres had disappeared considerably. This diffuse inflammatory change was found also in the rest of the cerebrum, and in the pons, bulb and cord. The pia was infiltrated with round embryonic cells, which, towards the base, at the bottom of the sulci, cluster into miliary nodules, as if the buds of miliary gummata. A third cranial nerve, infiltrated with similar cells, had undergone Wallerian degeneration.

In the cortex of some gyri were foci of œdema, visible to naked eye, *i.e.*, clear, rounded, more or less confluent spaces, their centres presenting a vessel, and in them the lymphatic sheaths and pericellular spaces dilated.

The vascular and meningeal lesion affects all the spinal cord, but especially the cervical, and the structure of the cord is masked by an amorphous exudate.

The larger arteries of the brain present slight patches of ordinary secondary obliterative endarteritis; but the chief lesion is an infiltration of embryonic cells in the adventitia, forming an intense periarteritis, diffuse, but in places nodular—the first stage of a gummatous process. The arteries of the cord are much less affected. In the veins the musculo-connective tunic is lesed; in them the infiltration, although usually diffuse, takes here and there a nodular character, even more clearly, and so as to form miliary gummata when "caseification" sets in. The venous lesion exceeds the arterial at the brain base—where it has led to hæmorrhage—and at the surface of the cord, and is largely independent of the meningeal and capillary lesion. The endo-vena does not show any proliferation; when it *is* affected it is so through invasion by the gummatous process. Hence the ulterior tendency of the luetic *arterial* lesion is to narrowing, obliteration, and their results; of the *venous*, to hæmorrhage.

Case 2.—Male, aged 42. General paralysis, with true tabes, affecting dorsal region most; and a superadded diffuse embryonic meningo-myelitis, almost exclusively affecting the posterior columns, and the cervical region most. In fresh cord, numerous granular bodies and some amyloid bodies in the posterior columns; a few of the former in the antero-lateral columns. In hardened cord:—*in posterior columns*; lumbar region, sclerosis of root zones and Lissauer's tract (slight or none in Westphal's marginal zone, Flechsig's oval centre and Strümpell's postero-external fields); dorsal and cervical regions, more extensive sclerosis of posterior columns. Bulbar sclerosis. In posterior columns, between some sound nerve fibres, are fibrillary tissue and granular bodies, and vessels also surrounded by the latter. Posterior nerve roots affected, especially the lower ones. In the posterior columns, arterioles also show diffuse embryonic inflammation, absolutely resembling syphilitic embryonic meningo-myelitis. Vessels of other columns show change of same kind. Meningitis slight.

Brain.—Vascular lesions of general paralysis, except in cortex of sulci of middle part of motor region, compared with which the superficial lesed parts of cortex show increased staining (hæmatoxylin) of nuclei of nerve cells, lessened number of nerve cells; increased number of neuroglia-nuclei and hypertrophy of its fibres; altered network of medullated fibres.

Case 3.—Male, 32. Old syphilitic transverse myelitis in upper dorsal region, with secondary degenerations, also with some thence-*ascending* myelitis of crossed pyramidal tracts. At base of brain, internal carotid and left Sylvian show endo-arteritis, thickened fibrous adventitia, and traces of embryonic infiltration. Capillary changes in brain cortex, precisely like those of cases 1 and 2, in all convolutions, more marked in motor and frontal regions. Meninges but little affected. No lesions of nerve-cells, or of tangential fibres, or of neuroglia. Slight alterations of sciatic, and of cutaneous nerves of hands and feet.

No mental symptoms, except three attacks of aphasia three years before death (connected with the left Sylvian and internal carotid lesion). Not general paralysis; but the promise of it in the future. The case is taken to show that in diffuse syphilitic meningo-encephalitis the vascular lesion precedes, and only afterwards are the nervous elements affected.

In an article in a recent number of BRAIN, I[1] have dealt very fully with general paralysis viewed as a disease of toxic origin, or of toxæmic origin (using either word in a wide sense); or, again, as a post-infection (*e.g.*, post-syphilitic) sequel, or degeneration of the nervous system, occasioned by the toxic influences chronically engendered subsequently (perhaps long subsequently) to infection; so that it is not necessary to add anything on that subject here.

Since the above was written, there comes to hand Dr. H. M. Bannister's recent interesting paper[2] on syphilis as directly causative of general paralysis; which last is held to be "a result of the action of a poison on the nervous system, and that poison in a vast majority of cases, at the least, a specific toxine. . . . . The toxine that produces the disease is in the system, and only waits for other conditions to prepare the way for its activity. Without these other conditions it is latent and may be innocuous."

B.—*Cerebro-spinal Syphilis: Invading Cerebro-spinal Meningitis.*

Syphilis often leads to a wide-spread gummatous infiltration of the cerebral and spinal meninges, which may be limited to the meninges, or also invade the substance of the brain, or cord, or both. It may begin at brain vertex, or at its base, or in the cord, or in brain and cord simultaneously. Therefore there may be, as syphilitic lesions, a specific cerebro-spinal meningitis, or a meningo-myelitis, or a meningo-encephalitis; and with any of these there may be gummata.

Tubercle and Syphilis.

Granulation tissue occurs in both; but—independently of bacteriological diagnosis—differences in site, the marked tendency of tubercle to caseate, may distinguish the two, in some instances. In syphilis the granular proliferation, or granular tissue, appears to begin in the smallest capillaries, either those of connective tissue, or nutrient of the larger blood vessels. The presence of the gelatiniform thickening of the cerebral or spinal meninges, and the absence of the minute tubercular granulations found in tubercular meningitis, may assist in indicating the syphilitic form.

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[1] BRAIN, Spring Number, 1894, pp. 61-4.

[2] *American Journal of Insanity*, April, 1894.

Syphilitic infiltration of meninges of brain and cord is a frequent form of syphilis; syphilis is rarely restricted to the cord; far more frequently is it cerebro-spinal.

This multiple cerebro-spinal syphilis, or specific cerebral and spinal meningitis, which also often forms special deposits or neoplasms in different parts of the central nervous system, or by proliferation invades the brain or cord, is usually evidenced, as Sachs[1] said, by the clinical association of:—

Symptoms of cranial nerve affections.

Spinal paralysis, most often taking the spastic type.

One or more great remissions.

Distinct history of syphilitic infection.

Invading, as it may, brain or cord, when, from the specific cerebro-spinal meningitis, the inflammatory process attacks the substance of the cord, the lateral columns are usually those first affected, and spastic symptoms therefore frequently arise. This syphilitic invasion apparently attacks the lateral columns most frequently, next the posterior columns, and the grey least often, and perhaps only in rapidly spreading meningo-myelitis. The base of the brain is attacked perhaps more frequently than the convexity. At first, before it is diffused, the process shows strong selective affinities, as, *e.g.*, the inter-peduncular space, the sixth nerve, and fibres for levator palpebræ; certain fibrils of nerves appear to be chiefly affected, and so (and by which alone) Sachs would explain certain partial ophthalmoplegias, occurring with or without other symptoms. But ocular palsies, internal and external, may not only follow this syphilitic inflammatory invasion of nerves, but also simple atrophy or simple primary degeneration of nerves, occurring as a sequel of syphilis, and occurring either singly or associated with other similar degenerative changes; and, as was indicated by Dr. Gowers,[2] these ocular degenerations may occur also, in inherited syphilis. Moreover, one must remember that the degeneration of tabes has been observed in congenital syphilis.

Of several of these cases by Sachs, the first was that of a married woman, aged 37, with a clear history of overt syphilitic disease.

Headaches, vomiting, vertigo, are soon followed by motor symptoms, ending in complete paraplegia; hiccough, constipation, retention of urine, photophobia, difficulty in speech. Under specific treatment, recovery so far as to be able to walk about and leave hospital. After a few weeks, re-appearance of general symptoms, headache, vertigo, vomiting, constipation, mental dulness; spastic paralysis, coming, by instalments, to affect all the limbs; unsymmetrical paralysis of various cranial nerves, incontinence of urine and fæces, atrophy of tongue, coma, death.

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[1] *New York Medical Journal*, September 19, 1891.

[2] "Syphilis of the Nervous System." Lettsomian Lectures, p. 48.

Thus *clinically* were—at first—chiefly a spastic paralysis of cerebro-spinal type; next, very considerable recovery; and, lastly, recurrence of spastic paralysis, eventually affecting all four limbs, and accompanied by paralysis of several cranial nerves; and *pathologically* were, at first, a general specific process affecting brain and cord; next, partly ceasing, but soon returning, and leading to specific formations in pons and cord.

At the brain base, were somewhat leathery thickening of pia at inter-peduncular space; swelling of optic nerves; constriction of other nerves by thickened pia, which was adherent to crura, pons and medulla oblongata. In the right half of pons, a gummatous lesion destroying right pyramidal tract, and invading left. Spinal dura very much thickened. Degenerated tracts in spinal cord.

Microscopically.—Typical syphilitic arteritis of vertebral and basilar arteries; their lumen much narrowed by a small-celled proliferation of the walls; in some places affecting the intima most, in others but little, while the adventitia was much thickened, showing cellular proliferation. In bulb and cord, degeneration of both lateral columns down to middle lumbar region, and of both anterior columns to lower dorsal region. Spinal pia adherent, thickened by marked round-cell infiltration. Special gummatous infiltrations here and there into substance of cord, at parts destroying cord substance from pia to degenerate area in pyramidal tracts, and to the grey. Gummatous lesion in pons, as above described. Blood vessels thickened and infiltrated.

Summary.—Syphilitic cerebro-spinal (chiefly lepto-) meningitis; with special invasion of pons and cord.

In the second case, the diagnosis was made from the entire history; the gradual development, first one side and then another being attacked; the remissions, and the almost complete recovery. The bilateral, but unequal, hypoglossal involvement, spastic paresis of both upper and lower limbs, and the marked remissions, tending towards recovery, "pointed to multiple foci of disease at the base of the brain, and probably in the cord also."

The third case was somewhat similar; the fourth was not of this type; the fifth was treated in an asylum for eighteen months, and mistakenly, as a case of general paralysis, but improved; in the sixth case the paralysis was flaccid (it was spastic in the others), and the knee and wrist jerks were absent.

Differential Diagnosis.—These cases of syphilitic cerebro-spinal meningitis, invading brain, cord, &c., are distinguished from insular sclerosis by the absence of intention tremor, of nystagmus, and of scanning speech; by the frequency of marked remissions; and, somewhat, by the history of syphilis.

From the tubercular form, they are separated by the less rapid course, and the greater tendency to wide-spread cerebro-spinal meningitis, and the lesser frequency of a limited basal or spinal meningitis.

From general paralysis, the diagnosis is often very puzzling.

A limited meningeal infiltration may simulate a tumour; nay, more, it may become one.

In syphilitic cerebro-spinal meningitis, some cases develop from above downwards, from brain to cord, as shown by brain symptoms first, and cord symptoms not until later on. But the cord may be first affected, and subsequently the brain; or both may, apparently, be attacked simultaneously. If the cord disease occasions paraplegia, and the brain disease hemiplegia, we have three-quarters paralysis, and such a form of paralysis, occurring by parts at different epochs, in *young* persons, is a tolerably significant indication of cerebro-spinal syphilis.

A case of cerebro-spinal syphilis with unusual cord lesions,[1] was that of a male aged 33. For about a year, severe headaches; visual disorders such as diplopia from paralysis of right external rectus, weakness of left superior oblique; and at times numbness on right side of face. On admission, two weeks before death, complete paralysis of left third and fourth cranial nerves (but right eye normal and no optic neuritis); hyperæsthesia of right side of face; right side of body hemiparetic; deep reflexes everywhere exaggerated. Coma preceded death.

Necropsy.—Many gummata involving both crura cerebri, left third and fourth cranial nerves, and right fifth, sixth, ninth and twelfth cranial nerves. Gummatous arteritis of vessels at brain-base. Small gummata in meninges and superficial substance of pons and bulb.

Lateral columns of cord paler than other white (in Müller's fluid). In lumbar enlargement, disease of anterior grey cornua, with hæmorrhages, disintegration, and cavitation, these changes varying in degree at different levels, and taken to be due to the lesion of blood vessels, and not to a primary poliomyelitis: the small vessels of the cord showed marked hyaline degeneration, and its meninges considerable small round-cell infiltration.

Many other cases of cerebro-spinal syphilis, owing to their important bearing on special points, are made use of in several other of the Parts or Divisions of this article.

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[1] H. M. Thomas, *Johns Hopkins Hospital Reports*, August, 1891.

C.—*Syphilitic Nervous Disease in the Aged, or those Above Middle Life.*

Cases of syphilis affecting the nervous system beyond middle life were reported by Mendel.[1] There is only space for the briefest summary of these.

Case 1.—Male, 60. Chancre; treatment by Hg.; cerebral symptoms culminating in unconsciousness; when this passed off, there were aphasia, hemianopsia and dextral paresis. These symptoms were attributed to syphilitic endo-arteritis of the Sylvian artery. K.I., recovery.

Case 2.—Wife of case 1. Faucial ulcers; painful swellings on parietal bones; contracted pupils, sluggish in reaction to light; weakened lower limbs. K.I., recovery, although for a time the headaches still came on occasionally.

Case 3.—Male, 59. Infected four years previously. Giddiness, facial palsy, left hemianæsthesia. Symptoms ascribed to left basilar arteritis. No change under treatment, except cessation of giddiness.

Case 4.—Male, 55. Condylomata; said to have had ulcer and rash a few weeks before. Cerebral hæmorrhage.

D.—*The Mental Symptoms of Brain Syphilis and Blood Syphilis.*

There are several sub-divisions which one might make of the mental and other cerebral symptoms due to syphilis, each of which offers advantages of its own.

For example, we may make a pathologico-anatomical division of mental and cerebral affections according as they originate from syphilitic neoplasms or from syphilitic inflammation of this or that intracranial structure, or from the luetic toxæmia, or from the luetic cachexia.

Or, when there is structural lesion of tissues, several groups may be formed according as the lesion chiefly attacks, or secondarily affects, either (*a*) the convexity of the brain and meninges; or (*b*) the arteries at the base, and the brain base itself; or (*c*) presents a clinical similarity to general paralysis of the insane.

Acute forms of insanity may come on in an early stage of syphilis, or in the middle and late stages.

At an early period there may be hypochondria; this may be interrupted or modified by supervening attacks of acute delirium, or of mania, or of melancholic type, and may gradually pass over into dementia. Or there may be persistent hypochondriacal and melancholic symptoms. Or the first very striking symptoms may be those of a mania, or of an active (or even acute) delirium (or even of expansive delusional disorder); which may alternate with mental depression, or with marked melancholic symptoms. Moral perversion sometimes supervenes on the acute mental disorders, and dementia does so pretty frequently. The acute or subacute mental symptoms may follow on epileptiform or apoplectiform attacks.

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[1] *Medical Press*, March, 1894, p. 310.

In some of the cases in which they occur, acute mental symptoms accompany the evolution of the secondary stage of syphilis, and especially its earlier periods. Here, mental depression, or active mania, or delirium, may come on early, and often quite independently of any convulsive or other severe nervous affection dependent upon syphilis. Yet at this early date there may be other nervous symptoms produced by syphilis, such as convulsions, severe headache, insomnia, anæsthesia, neuralgia, facial paralysis, hemiplegia; and these conditions may exercise a modifying influence on the morbid mental derangements in question.

In such cases the acute or subacute mental symptoms are attributable, often, to the syphilæmia following the primary infection, and, as I suggested long ago, "the blood change produced by the syphilitic virus probably acts much as a dram of spirits acts upon the hereditarily weak brain in producing a temporary *mania a potu.*"

On the other hand, as the present writer also stated, "considerable maniacal excitement, or even acute frenzy, may occur in the course of mental decay or derangement produced by intracranial syphilitic lesions of the *late stages*. . . . . In many cases such as these the outbursts of acute mental symptoms follow upon convulsive seizures caused by organic syphilitic disease of the tertiary type."

At the same time, stating in summary the symptoms in ten cases of intracranial syphilis with insanity—cases in which, therefore, the stage of syphilis reached was a late and not an early one—the present writer drew attention to three great groups of symptoms, or clinical features, as being especially worthy of notice in these and similar cases. My summary was as follows:[1]—

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[1] *Brit and For. Med. Chir. Rev.*, Oct., 1876, p. 440.

"The first is the marked tendency to mental impairment, failure, or dilapidation; the predominance of negative intellectual symptoms, often associated with failure, weakness, or loss of the moral sense, of the self-regarding virtues, and the general inclination to a degraded state of feeling, as evidenced by the habits of the patients. In some, early or intercurrent outbreaks of maniacal symptoms occurred; in a few there was causeless depression or fear; or emotional weakness, as displayed either in a lachrymose tendency or in a disposition to uncalled-for merriment.

"The second broad clinical aspect of importance . . . is that almost invariably there were motor symptoms of the paralytic or of the convulsive type. In the former—the paralytic—type, motor failure was in the form of hemiplegia, or palsies of cranial nerves, or impairment of articulation, or motor paresis and incoordination of a widespread kind, or impaired innervation of visceral organs. The latter—the convulsive—type of symptom showed itself more particularly in epileptiform attacks, hemispasm, general convulsive seizures, tremors, or symptomatic paralysis agitans.

"The third general feature is that sensory symptoms were frequent. Of these, the most striking was intense nocturnal pain, especially cranial pain, while anæsthesia was far less frequent. Nor did the special senses escape, for impairment of sight, blindness, or unilateral deafness were occasionally found. Rarely were hallucinations of the senses observed."

In other cases, in which there is not decided evidence of gross local intracranial syphilis, the mental symptoms are often of a more active form than those above mentioned, although perhaps occurring at a considerable interval after primary infection. Of these, brief references to some of the examples I[1] have published follow:—

In one case, within a year of primary infection mentally affected, there were, successively, primary and secondary syphilis, cranial pain, alopecia, return of cranial pain, breach of discipline when under the influence of drink, imprisonment, intense cranial pain, worse at night, insomnia; stupor and delirium, worse at night, and alternating or succeeding each other irregularly.

He was violent in temper, easily angered, of impaired memory. Overt insanity took on the form of mania of an acute or subacute type, subsiding eventually into a quiet expansive delusional state. Recovery under specific anti-luetic treatment.

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[1] *Journal Mental Science*, Oct., 1879, and Jan., 1880.

Another case, with a history of protracted severe constitutional syphilis, had an attack doubtfully attributable to sunstroke; and, later on, hepatitis, general debility, dyspepsia, cranial pain, vertigo, insomnia, failure of moral sense, dementia, tinnitus aurium, some anosmia and ageustia. Probable specific pachymeningitis and vertical leptomeningitis, with organic changes in cerebral cortex and arterioles. Recovery under antisyphilitic treatment.

A third case presented the following succession of events. Constitutional syphilis, rash, convulsive seizures, mental depression, hallucinations of sight and hearing, delusions of conspiracy against him and designs against his life; periostitis of tibiæ, sternum and cranium. Subsequently, pericranitis, severe nocturnal, tibial and cranial pains, insomnia. Attacks in which the delusions and hallucinations became vivid, with tenderness and intense pain at the forehead and vertex, worst at early morning, and dissipated by large doses of potassium-iodide. Several ague-like paroxysms. Scaly coppery patches on calf of one leg. Mental depression and impairment, hallucinations of sight and hearing, delusions of conspiracy and persecution, under which occasionally came excitement or even violence. Recovery.

In a fourth case were a history of primary and secondary syphilis, and subsequent general "rheumatic" pain; later on, mental depression, suicidal attempt and tendencies, hallucinations of sight and hearing, delusions as to designs on his life by poisoning, and as to his incessant torture at night by means of mysterious agencies, and carried out by persons who followed him everywhere with that object. Impairment of memory, and of other mental powers. Later on, he alleged that clocks ticked in his head, and that voices and trumpets were blown into it. Jaundice, severe and prolonged cranial and chiefly nocturnal pain, with local tenderness, relieved by specific treatment. Hepatic disease; incipient phthisis. Recovery; probably temporary.

A fifth case; whilst under treatment elsewhere for so-called "rheumatic" pains, but probably syphilitic in origin, and associated with anæmia and cachexia; took meningitis, and subsequently melancholia with hallucinations of hearing, defective memory, and some degree of mental confusion. Latency of external syphilis. Cerebral and general anæmia. Later on, syphilitic cranitis, pericranitis, and pachymeningitis, slight local paralysis (face, leg); hemiplegia and vomiting (following a node on the cranial vertex, and pain in the limbs). Localised and partly nocturnal cranial pain. Died of another disease. At the *necropsy*, were, *inter alia*, syphilitic ostitis ending in caries and necrosis of skull, syphilitic pericranitis, and pachymeningitis. Also traces of gummata of liver.

A sixth patient had had, at the onset of mental disorder, an attack of stupor and of maniacal excitement with tendencies to acts of violence, followed by maniacal symptoms such as restlessness, &c., and paroxysmal excitement, rapidly deteriorating into profound dementia. Improvement under specific treatment. Later on, recurring pericranial nodes, and intense cranial pain sometimes worse at night, with mental dulness and insomnia. Ozœna. Atrophied testicles, which were irregular in shape, as if scarred, or partly in fibroid change. Irregular tibiæ, coppery psoriasis on calves of legs.

Death from another disease. *Necropsy;* of the lesions present, those assigned to syphilis were old internal and external cerebral pachymeningitis, partial leptomeningitis; and perhaps, the traces of perisplenitis found.

In one case by Belkowsky,[1] the several successive medical attendants of a (supposed syphilitic) woman aged 41, diagnosed "Nervous excitement," "Neurasthenia," "Hysteria," "Insanity," "General paralysis."

In his work on brain syphilis, already cited, Prof. Fournier both divides the cerebral symptoms generally, and sub-divides the groups thus formed, including the mental, in an over elaborate way. But here will be added only a portion of a summary and critique, already published by the present writer, on the portion of Fournier's book which deals with mental disease from syphilis. Curtailed, it was as follows:—

The symptomatological forms of brain syphilis are either:—

A.—The early, the initial, the primary; or

B.—The consecutive, the terminal.

As for the former ("A") Fournier divides them into six forms; (1) the cephalalgic; (2) the congestive; (3) the epileptic or convulsive; (4) the aphasic; (5) the mental; and (6) the paralytic.

These forms only represent groups of symptoms, several of which almost invariably are combined, and in the most varied ways, in actual clinical experience. And an analogous criticism applies to the sub-divisions made of the fifth—or mental—of these forms; the one to which attention is restricted in what follows.

The intellectual troubles of cerebral syphilis pertain to two great groups; the one (I.) consisting of symptoms slow of evolution, and of depressive form, constituting a kind of gradual sinking in intelligence, of mental lowering, of progressive hebetude. The other (II.) consisting of symptoms more active, of more rapidly developed intensity; viz., of the phenomena of cerebral exaltation or excitation, of more or less acute states of raving, of mental derangement, of mania.

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[1] *Centralblatt für Nervenheilkunde and Psych.*, April, 1894, p. 169.

I. Of these groups the former "I." is by far the more frequent. According to Fournier, it allows of two degrees or varieties :—

(1) Simple intellectual enfeeblement.

(2) Incoherent hebetude, or intellectual failure with incoherence.

In the former of these varieties the intellectual impairment is associated with changes of disposition, habits, and temper. The most remarkable of the symptoms is the greater or less degree of amnesia—amnesia the oncoming of which is either gradual and progressive; or is attained, as it were, by leaps; or rarely, by a sudden "amnesic stroke."

On the other hand, in the latter of the above varieties of group "I.," mental failure and mental alienation co-exist, and may do so from the outset; but in the vast majority of the cases the symptoms at first are merely those of simple intellectual weakness, which, later on, become complicated with insanity. So that, to the intellectual enfeeblement of the former variety, there is here superadded an intellectual disorder or derangement; of this latter, incoherence is the predominant expression.

Finally, in a few exceptional, indeed rare cases, the mental disorder is more partial, more systematised.

II. The second large group "II." of intellectual troubles, taking origin in cerebral syphilis, is constituted by phenomena more active than these observed in the first group "I.," more rapid in their invasion; and consisting of mental excitation; or delirium; or maniacal incoherence; or mania. Or, far more rarely, consisting of vivid hallucinations; or acute melancholia; or persecutory monomania; or impulsive insanity.

Formerly, at least, Fournier failed to distinguish the cases of acute syphilitic insanity supervening during the early secondary stage of the evolution of syphilis, from those supervening during the tertiary stage. Yet they must be kept distinct, their pathogenesis often differing.

The more active mental disorders of the group just described, and dependent upon cerebral excitation, follow one or other of two modes of invasion. For they may be preceded or accompanied by other cerebral affections of specific origin, whether headache, convulsion, aphasia, or seizure apoplectiform or paralytic; and these associations and combinations of various cerebral symptoms are by far the more frequent; or, on the other hand, the psychoses in question may be isolated, and free (at least at first) from any such association or combination; and this is much the more rare mode of invasion.

In the former class the diagnosis of the specific nature of the case is relatively easy. The variety, association, and succession of the symptoms reveal the specific nature of the phenomena, and establish the diagnosis on a rational basis.

In the latter, and more rare class, that in which the intellectual disorders are neither preceded nor accompanied by other syphilitic cerebral affections, but occur in single and solitary isolation, the diagnostic difficulties are far greater. No history of syphilis may be obtainable, and if so, the difficulty in diagnosis is at its maximum. But, granted a history of syphilis, and, later on, the advent of mental disease—what warrants us in attributing the latter to the former as effect to cause?

Is it some particular type or pathognomonic attribute of the mental disorder?

Is it the detection of co-existent indications of syphilis in this part or in that?

Is it the verification of personal or hereditary predisposition to psychoses?

None of these will be our warrant. For the types are the same as those of mental disorders from other causes; contemporary signs of non-nervous syphilis are usually wanting; and, for obvious reasons, the presence or absence of predisposition has only a restricted and comparative value. Thus, in the life-history of the same subject we are reduced to the two facts—syphilis and mental alienation—occurring in successive chronological order. Hazardous speculation and assumption supplant legitimate conclusions as to the pathogenetic relationships of the mental alienation when its syphilitic origin is postulated, in such case.

Nevertheless, all such cases—unless clearly *not* of specific origin—should be submitted to careful tentative anti-syphilitic treatment.

E.—*Cephalalgia and Insomnia in Intra-cranial and Cranial Syphilis.*

Cephalalgia is one of the best authenticated, simplest, and most frequent modes of expression of the onset of cerebral syphilis. In its various degrees, and singly, or associated with other manifestations, it was believed by Fournier to usher in cerebral syphilis in about two-thirds of the cases. The pain he stated not to be neuralgic nor rheumatic. It is deeply seated; to the sufferer it seems to be within the head, or within the brain; an encephalalgia. Most often, it is either of heavy, or of constrictive, or of hammering character. Sometimes it is straitly circumscribed in site: sometimes diffused over a region, or several regions, of the brain; rarely over the whole head.

The three principal characters, or significant peculiarities, which testify to its luetic nature, are:

(1) Its great intensity; and sometimes extreme violence or frightful severity, so that the sufferer writhes in agony. Transitory delirium may accompany the more severe degrees of it.

(2) Its nocturnal exacerbations: the pain sometimes is very notably worse at night or evening. In some cases the pain is but little increased at night, in a few cases it is less by night than by day. This last is far more likely to occur in tertiary syphilis, in which, indeed, the headache is more continuous and equable than in secondary. Hand in hand with the nocturnal pain goes insomnia.

Valuable, therefore, as is the nocturnal exacerbation of the headache in cerebral syphilis, it is not a constant sign; indeed, it is not seldom absent.

(3) The third character, in evidence for the luetic origin of the headache, is its long duration and relapses.

As Fournier long ago stated, an intense or violent cephalalgia, with nocturnal exacerbations, of long-standing, and frequently relapsing, almost infallibly testifies to syphilis. But it is not always present in brain syphilis.

It is a most important phenomenon as a premonitory symptom, other cerebral symptoms of syphilis often following in its train. It often forms, so long beforehand, a prelude of the coming deadly evil, as to enable prompt and vigorous treatment to ward off the latter, if the warning be heeded and the occasion's opportunity embraced.

From the characteristic syphilitic cephalalgia, is to be distinguished that which is a chief symptom of the so-called "neurasthenia of syphilitic origin," relatively often found at the beginning of the second stage of syphilis, and especially in young women predisposed thereto. The clinical aspect is as varied as is that of ordinary neurasthenia, and, as in the latter, hysterical symptoms often accompany.

Yet neurasthenia of syphilitic origin may come on much later, say from two to seven years after infection. Neurasthenia occurring in the late stage of syphilis takes two chief forms;[1] one, the more frequent, abortive or incomplete, as neurasthenic headache; the other, the rarer, as a complete form of cerebrasthenia or myelasthenia.

In either form, the neurasthenic headache is a prominent feature. It is an indefinite morbid feeling in the head, described as a heaviness of head or feeling of compression there. From the peculiar headaches of syphilis, in the second and third stages, already described, these neurasthenic head-pains (of syphilitic origin) differ by being much less painful, by not undergoing nocturnal exacerbation, by the little effect on them of antiluetic treatment, and by their long duration.

Headache of syphilitic nature, in its ætiological relations, may roughly be divided into two forms: the one being headache with lesions (of bones, meninges, &c.); the other being headache without such lesions, but of purely nervous character, and the outcome of over-irritability of brain cells. The first form requires specific treatment. Iodide is the best; but, exercising its curative effect gradually only, it must for a time be supplemented, in severe cases, by narcotics, of which chloral gives, perhaps, the best results. For the second form, antipyrin is most to be eulogized, and iodide yields some satisfactory results.[2]

"Syphilitic" headache the *direct* outcome of lues, is, I think, in some cases due to specific syphilitic processes affecting brain, meninges, nerves or skull; and in some cases is the neuralgic expression of syphilæmia.

Moreover, various forms of headache, as for example the neurasthenic, may be the *indirect* outcome of lues.

In many cases we have nothing except the history of chancre or of sequelæ, and perhaps the effect of treatment (but only if its *success* is striking), to guide us in diagnosing the syphilitic nature of a brain disease. But Dr. L. C. Gray[3] ventures to outline what he takes to be a pathognomonic symptom-group of brain syphilis, in one set of cases; "namely, a cephalalgia that is apt to be peculiar in a quasi-periodicity that manifests itself in a tendency to return at a certain time in the twenty-four hours, most frequently at or towards night, less frequently in the afternoon or morning; marked insomnia, usually at the outset, lasting a few weeks; a sudden cessation of the cephalalgia and insomnia upon the supervention of any paralytic or convulsive symptoms." His view, expressed in this connection on hemiplegia, is practically that so long held by many. He gives outlines of twenty-seven cases presenting that symptom group, and tabulates them by selecting prominent symptoms. Treatment was satisfactory in many of the cases. The insomnia and cephalalgia generally belong to the early stage of intracranial syphilis; but may be found at any stage "of the general syphilitic infection." These two co-existent symptoms he has met only in one non-syphilitic case—one of acute bulbar palsy.

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[1] Fournier, *Le Bulletin Médical*, 1893, Nos. 80-82.

[2] Lionnet, *Thèse de Lyon*, 1893.

[3] Read before the American Neurological Association, September, 1891.

Now, this cephalalgia and insomnia, so long well-known, are commonplaces in syphilitic nervous disease, and are very often the only symptoms for which the patient, totally unconscious of their luetic origin, seeks relief. Nor do they by any means always disappear on the supervention of any paralytic or convulsive symptoms; for they may be, and remain, excessive, with, for example, hemiplegia of luetic origin. But why erect this symptom-group, of two only, into a *pathognomonic* sign or symptom? And the doing of this is the one new point in the article.

The cephalalgia and insomnia are ascribed to gummatous infiltration of the meninges; for the pathological changes of cerebral and cerebellar substance do not usually occasion pain, except when these organs are the site of neoplasms, and not always then. And the striking effect of vigorous anti-syphilitic treatment in cases with only the peculiar headaches and insomnia, is taken by Gray to give support to that ascription.

PART II.

SOME ASPECTS OF CONGENITAL OR HEREDITARY SYPHILIS.

A.—*Some Clinical Aspects, Relationships, and Laws of Congenital Syphilis.*

Certain dogmatic assertions, or so-called laws, hitherto generally accepted with regard to congenital syphilis, are contested by Dr. Albrecht Erlenmeyer,[1] in consequence of the light shed upon the matter by some clinically observed examples, which more immediately concern the department of late or tardy hereditary syphilis; to which subject we may now turn attention;

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[1] *Zeitschrift für Klin. Med.*, Bd. xxi., H. 3, u. 4.

as well as to description of a form of congenital syphilis in young persons; and to the relation of congenital syphilis to some other brain and nerve diseases.

Tardy, or late Hereditary Syphilis.

Case 1.—A syphilitic father died of brain syphilis. A son of his, born two years after the father had incurred the specific disease, did not develop signs of congenital syphilis until his twelfth year, and then in the form of ulceration of pharynx, a tolerably wide-spread squamous rash on chest and nape, and general swelling of glands. Recovery under treatment. The mother has not manifested any indications of syphilis.

Case 2.—A father, infected before marriage, died of syphilitic disease of the cervical spinal cord and membranes. The second of the sons, in the thirteenth year of his life, exhibited the first signs of congenital syphilis, affecting bones and joints. Recovery under treatment.

This tardy or late hereditary syphilis has been recognised by Virchow, Weil, Sevestre and others; but denied by Edm. Lesser.

The upholders of the doctrine of tardy hereditary syphilis maintain that, precisely as in the acquired form, so, in the congenital, may the syphilitic virus remain long latent in the organism, until aroused by some excitant conditions, not yet sufficiently known or understood as to their quality and extent. Thus, manifestations of *hereditary* syphilis, hitherto latent, may be roused by the rapid growth of childhood, the cataclysmic changes of puberty, febrile disease, injury; to which the present writer would add drink (not unknown in early life) and masturbation.

On the other hand, *acquired* syphilis is more frequently driven out of latency by alcoholic or traumatic influences.

The doctrine that the mother of a child inheriting syphilis from the father, is never infected by suckling the child, whereas a non-syphilitic nurse is infected on the breast by suckling the same child, is not true in all cases.

Case 3.—A luetic man married a healthy woman. Abortion ended the earliest resulting pregnancy. The first child that was born alive was a girl, was luetic, and died in two weeks. The second child born alive was a boy, and was healthy; the third born alive, also, was a boy, and healthy. The fourth, and last, was a girl, and luetic. The mother was not syphilitic herself, at any time.

Cases such as these, and the more or less similar forty-three examples of Kassowitz, prove that a healthy mother, bearing a congenitally syphilitic (*per patrem*) child, *may*, herself, remain uninfected with syphilis, although, of course, she very often, or almost always, *is* infected. Several cases have shown the following sequence of events:—A syphilitic man marries a healthy woman; abortions and syphilitic children come; the mother remains uninfected, the father dies of brain syphilis. Those who hold that the father transmits syphilis to the offspring only by the mediation of syphilitic sperm cells, can also readily accept the statement that the mother sometimes is not infected in such cases.

The so-called law of Colles is against this view; namely, the law, doctrine, or dogmatic assertion, that the mother bearing a syphilitic child is never infected (at the breast) by suckling it, whereas a healthy, *i.e.*, non-syphilitic wet-nurse is apt to be infected, and in that way. This law involves acceptance of Ricord's doctrine (that, so-called, of "*unicité*"), to the effect that syphilis can only be incurred once—one true infection making immune against future fresh contraction of the disease. Thus, if Colles's law be accepted, one must argue that because the mother, suckling her syphilitic child, gets no breast syphilis, she is immune from syphilis, and because she is immune from syphilis she is thereby proved to be already infected and syphilitic. But indubitable examples of a second syphilitic infection have been observed by Zeissl, Diday, Köbner, Bäumler, and others. Implicitly, in Colles's doctrine the conceptions "syphilitic" and "immune from syphilis" are taken, and erroneously so, as having the same significance, practically. As well say that the vaccinated person is immune from variola because already variolised, and in such sense as to be capable of infecting non-immune persons with variola.

Moreover, in Case 3, above, a striking and significant fact is that we find only the two girls congenitally syphilitic (following the luetic father); the two boys escape (following the non-luetic mother). And not only so, but one syphilitic girl is born *before*, and one is born *after*, the two boys exempt from syphilis.

Case 4.—The fourth case presents somewhat similar features to the third case as regards the sex of the children, except that here the first child soon died, but without signs of syphilis; the second, a girl, was healthy; the third, a boy, was syphilitic.

Since the above was written a case is reported by Dr. J. A. Coutts[1] as being in contradiction with Colles' law. In this case a mother, suckling her syphilitic infant, whose mouth, tongue and lips were among the parts showing luetic lesions, incurred primary syphilis of the breast, in due time followed by typical secondary syphilis. But it is possible that the infant's syphilis was acquired and not hereditary.

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[1] *The Lancet*, June 9, 1894, p. 1443.

Colles's law goes to support the view that syphilis in the fœtus is always of maternal origin; the law, namely, that a mother, apparently healthy, is never infected by suckling her syphilitic infant, even if her nipples be fissured or sore, and the infant's mouth be affected with contagious syphilitic lesions or rash: whereas a healthy wet-nurse suckling the same infant, when she has sore or cracked nipples, will incur a syphilitic sore followed by general syphilis; and this immunity of the mother to syphilitic infection is attributed to her being already syphilitic, even if she presents absolutely no other indication of being syphilitic.

Schuster, of Aix-la-Chappelle, has recently discussed some matters connected with this subject, a subject which presents many points of practical importance.

Thus, it has been held that where a husband and wife are non-luetic, and the wife becomes pregnant by him, and the husband, subsequently acquiring syphilis, infects his pregnant wife, the latter takes a primary sore, followed by what I will call *syphilæmia*; the fœtus gets no primary syphilitic lesion, but the virus is conveyed to it by the blood of the mother. And moreover, that the result usually differs with the point of time in the course of pregnancy that the mother takes the primary infection, inasmuch as "the virus takes a period of eight or nine weeks to enter the system of the mother, to make her constitutionally syphilitic, and to infect her blood with the syphilitic virus."[1] So that, if the mother takes the primary infection during the seventh month of pregnancy, or later, the fœtus will probably remain free; whereas, if the mother incurs the primary luetic infection at an earlier point of time during the pregnancy than the seventh month, the infection (usually, at least) passes from mother to fœtus, the germ of syphilis being communicated to the child through the mother's blood by the connection between placental and fœtal circulation, the placenta not forming a barrier to infection, but, on the contrary, sometimes becoming the site of syphilitic morbid processes itself.

In reference to the above I must confess that it seems to me improbable that the fœtus should escape if the mother became infected "during the seventh month of pregnancy"; or that eight or nine weeks must elapse before the luetic virus infects the mother's blood

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[1] *Provincial Medical Journal*, August, 1893, p. 437.

Furthermore, the above Case 4 by Erlenmeyer is in opposition with the law or doctrine of Kassowitz. Likewise does Case 5 contradict Kassowitz's law; namely, *the law of the spontaneous gradual weakening of the intensity of syphilitic inheritance*; so that the nearer conception is to infection, the more certain is inheritance of syphilis, and the later born children have a better chance of escape; and that, finally, hereditary transmission fails and ceases. As just stated, the two cases (4 and 5) are in opposition to this law or doctrine. So also is the statement of Coutts that he has "seen several cases of twins where one was deeply affected, and the other seemingly healthy." He also refers to the possibility of birth of a syphilitic child both before and after the birth of intermediate healthy children, in the same family; in this resembling Cases 4 and 5, therefore.

B.—*A Form of Congenital Brain Syphilis.*

One form of congenital syphilis is manifested by unilateral epileptiform attacks in the limbs, at first following a febrile attack. On the side thus affected by convulsions from time to time, the limbs become shortened and lessened in size, but although therefore weaker than those of the other side, do not show paresis proper, nor spastic conditions, nor altered electrical reaction of muscles or of nerves. Yet they show some hyperæsthesia, especially of kinæsthetic nature, an impairment of the feeling of localisation of the part, lessened consciousness of the position of the limb; and, also, of the loading of it with different weights. The extremities on the side affected are short and slender, in consequence of a restraint of growth referable to an impeded, restrained development of the cortical brain centres representing those parts. Some cases have such symptoms as hemiatrophy of tongue, ptosis, facial paresis, visual atrophy; some cases have a history, pointing to meningitis or peri-encephalitis in infancy (as indicated by the tale of teeth-grinding, squinting, stiff nape, and of the treatment employed).

The conclusions of Erlenmeyer are:—

Hereditary syphilis may be tardy. It may remain latent even beyond the age of 12 years.

Puberty, injury, and febrile diseases, are effective excitants in making manifest a hitherto latent congenital syphilis.

The doctrine of Colles has no absolute validity, inasmuch as whilst mothers of syphilitic offspring may have become syphilitic that does not necessarily occur. And, moreover, "immune from syphilis" is not equivalent to "syphilitic."

The doctrine or law of Kassowitz is not always applicable, for, after infection, the later born children may be more severely infected than the earlier born ones.

The incidence of hereditary syphilis may be on the children of one sex only, in the family. If, after the birth of a syphilitic infant, a child of the opposite sex is born free from syphilis, that does not show that the capability of inheriting the paternal syphilis is extinct.

Anti-syphilitic treatment of the parents has a more favourable influence on the later born children.

A form of cerebral disease with unilateral arrest of development and convulsions, in children, is mostly due to congenital syphilis.

The so-called cerebral paralysis of children frequently pertains to congenital syphilis.

Congenital epilepsy, with or without idiocy, very often is due to inherited syphilis.

C.—*Congenital Cerebro-spinal Syphilis.*

Although frequent in adults, there are comparatively few cases on record of congenital syphilis of both spinal cord and brain, particularly if attention be limited to the examples microscopically examined. Congenital cord syphilis, without brain syphilis, is scarcely known; at least is rare.

From Professor Westphal's clinic, an example of congenital brain and cord syphilis was minutely reported by Dr. E. Siemerling,[1] with necropsy and microscopy.

Gummy encephalo-meningitis; basilar gummy arachnitis; internal hydrocephalus; gummata of dura mater; atrophy of the dura mater of the convexity; atrophy of calvaria; multiple osteoporosis of the skull-base; spinal gummy arachnitis.

The patient, aged about 12 or 13 years at death, child of a syphilitic father, at the age of 4 years had a unilateral stroke with paralysis of right limbs, and loss of speech; the latter was soon regained, the former only gradually lessened, so that the right arm was weak a year later. About two years after this attack visual power diminished, and by ophthalmoscope was seen commencing atrophy of optic discs. Owing to weakness of the legs, the child was in hospital for a short time; ataxy affected all the limbs. Sensory disorders were not observed.

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[1] *Archiv. fur Psychiatrie*, Band xx., Heft. 1, p. 101.

At the age of 11 years, the child quite lost sight, and in place of attending an ordinary school, as had hitherto been possible, had to go to one for the blind. But the mental powers were not yet observed to be deranged or impaired.

Eleven months before death, severe symptoms were again observed:—vomiting, constant headache, attacks of vertigo. Three months still later, there began genuine epileptiform seizures with loss of consciousness. Walking was extremely difficult, fatigue very easily induced, severe vertigo complained of.

Three months still later, she almost entirely lost the power of hearing. One month later, and therefore, four months before death, were almost complete bilateral deafness, bilateral optic atrophy following neuritis; enlarged hydrocephalic head, painful on movement or percussion; unsafe, ataxic gait, with vertiginous feeling; sensibility difficult to test, but that to pain existed. No appearance of paralysis; no note made of mental anomaly or of abnormal knee-jerk.

Subsequently, were repeated severe convulsive attacks, chiefly tonic spasms, sometimes with extreme beclouding, sometimes with loss, of consciousness. Usually with these were evacuation of urine, slow pulse, and afterwards hunger and languor; vomiting also occurred after these attacks, or else independently of them; twice in the attacks, were slight right ptosis and facial paresis; once, slight dextral hemiparesis. The gait became gradually worse, the vertigo so marked that the patient could scarcely hold herself up, even in the sitting posture. The gait was always ataxic. At four different dates, within three months or so, the knee-jerks were alternately present and absent.

Finally, were states of transitory somnolence. From being mentally clear and alert, the patient at times became peevish and morose. Increasing, the spasmodic seizures ushered in death.

At the *necropsy* were internal hydrocephalus, large gummatous growths, chiefly at the brain-base, which had damaged the parts here, including the cerebellum, and the nerves issuing from them. The tumours were of syphilitic granulation tissue, taking origin in the soft meninges of the brain. In the walls of the blood-vessels were syphilitic changes. The syphilitic infiltration had led to complete degeneration of some nerves (*e.g.*, the optic), to less of others (*e.g.*, the oculo-motor); the new growth affected the pons and bulb (M.O.) most, and chiefly their ventral aspect, but a portion of it from the cord projected into the fourth ventricle.

Throughout the spinal pia (especially at middle and lower part), was marked gummatous growth, invading the substance of the cord itself irregularly, with here and there peg-like projections. Almost throughout the cord were the posterior columns lesed, partly by direct infiltration, partly and secondarily by pressure; and similarly these two processes co-operated in destroying the nerve fibres affected; most of the nerve *roots*, however, were exempt, or nearly so, from change.

In the dorsal part of the cord, the lateral portions also suffered severely.

The vessels of spinal pia and white substance showed the obliterative phlebitis described by Greiff[1] in a case of spinal cord syphilis. The spinal arteries showed less alteration than the cerebral.

The cerebral symptoms: atrophy of optic nerves, spasmodic and convulsive attacks, vertigo, vomiting, deafness, transitory palsy of some cranial nerves (*e.g.*, oculo-motor and facial), are accounted for by the hydrocephalus, by the syphilomata, &c., and the implication of nerves.

The spinal symptoms are less clearly accounted for; with the pathological spinal changes found, more marked clinical features might have been expected. The parts most affected were the posterior columns, but these only slightly so from lumbar swelling downwards; the nerve roots were mostly spared. The ataxy observed early in and throughout the course of the symptoms may be explained by this posterior columnar change; or by this *plus* the basal intracranial new formation, particularly that of the cerebellum. Lancinating pains were absent; the state of the knee-jerk was variable; at dorsi-lumbar junction, the posterior root zone was slightly affected on the left side, part of it affected and part exempt on the right. Sensory disorders were difficult to make out.

D.—*Organic Hemiplegia following Slight Injury in Hereditary Syphilis.*

A boy, aged 6 years, immediately after a fall from a child's go-cart, presented the classical symptoms of right organic hemiplegia, without loss of consciousness and without aphasia. The father of the lad had been ten years syphilitic, was of the arthritic diathesis, and suffered from renal colic. The mother was nervous and neuralgic. The boy himself was born two months before term, and was slow in teething and in learning to walk. At the age of 6 years, when his accident befell, he was strumous, liable to ganglionic engorgements, bled easily on slight abrasion, and had frequent urticarial eruptions. He presented various stigmata of degeneracy in conformation of ears and palatine arch. Some teeth were irregular and notched.

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[1] *Archiv. fur Psychiatrie*, Band xii., p. 565.

He fell on the right side, and mainly on the point of the shoulder. No loss of consciousness attended this little accident; the paralysis followed it immediately, and when examined seventeen hours afterwards by Dr. Paul Blocq,[1] there were no external traces of injury, no mental symptoms; a little defect of speech was due to the facial paralysis; otherwise he answered and read well, and had no aphasia.

The only deviation from the normal was a total hemiplegia of the right side, more intense in the upper limb, less in the lower, little marked in the face, and affecting the trunk. As to the *face*, there was some difficulty in raising the right eyebrow and lid, nor could he grimace at will on that side, although in laughing both sides moved. Mastication was a little impaired by the cheek paresis. No deviation of tongue. No other motor disorders. Sensation, sphincters, reflexes and temperature, all normal or nearly so. The lad was cheerful and slept well.

Thus, there was a right motor hemiplegia (limbs, lower face, trunk), suddenly following a slight fall, and not accompanied by loss of consciousness.

Of what nature was it? Was it a hystero-traumatic hemiplegia, which would be the first question to arise in one's mind? That it was *not*, was indicated by the absence of hemianæsthesia of hysteric type, by the absence of all kinds of sensory disorders; by the failure to discover hysterogenic zones, hysterical stigmata of other kind, or previous hysteric manifestations. The hemiplegia itself, with the characters of the organic form of hemiplegia, told against that hypothesis; for in the face it affected the lower part only (and without spasm); of the limbs, the upper more than the lower, and in the limbs the extremities more than the proximal parts. In the same direction, told the cheerfulness, gaiety, and normal sleep.

Was it an infantile spinal paralysis? That it was *not*, was indicated by the absence of symptoms affecting the general state, by the apyrexia, the onset after slight injury, the normal tendon-reflexes.

Concluding the hemiplegia to be "organic," what was the *site* of the lesion producing it? Its onset without apoplectic shock, without even slight stunning, showed the central lesion to be of little extent, whilst, on the contrary, the degree of intensity of the paralysis (or paresis), and its distribution over the whole

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[1] *Revue Neurologique*, January 30, 1894.

right half of the body, showed participation of a large portion of the motor zone. This conjunction indicates the anterior part of the posterior segment of the left internal capsule as being the site of the lesion. A meningeal or cortical lesion, sufficient to produce the hemiplegia present in this case, would doubtless have occasioned other symptoms, not present.

What was the *nature* of the lesion?

The sudden onset, without prodromic symptoms, excludes brain-inflammation (polio-encephalitis) or a neoplasm; nay, it points to a vascular disorder. The fall was such as almost to preclude the possibility of rupture of a healthy blood vessel, and as to make a pre-existing fragility of the vessels appear to be indispensable for the occurrence of rupture.

The child had a predisposition to urticaria; was the hemiplegia due to a vascular disturbance of this order in the brain? He had also a tendency to easily produced and undue hæmorrhage—a tendency to hæmophilia. Might this, under the excitant slight shock of the fall, have led to a capillary hæmorrhage sufficient to give rise to the hemiplegia? These are possibilities; but, on the other hand, there are the more suggestive facts of paternal syphilis, and of the premature birth and dental anomalies of the lad, which render it probable that the vascular disorders were directly or indirectly promoted or engendered by a predisposition, originating in hereditary syphilis.

The recovery began before the specific treatment was adopted, and in a fortnight was far advanced.

The syphilis probably acted indirectly, inducing general defects of development, and bringing about morbid fragility of blood vessels, whence a tendency to urticaria and to hæmorrhage, the latter occurring easily on slight injury, and on this occasion giving rise to hemiplegia.

Of course, syphilis in children is not always hereditary, it may be *acquired* by them in various ways. The last I have seen reported, and since the above was written, were several cases,[1] the experience of the reporter of which is that the secondary symptoms of acquired syphilis in children are more severe, as a rule, than in adults, and syphilis less amenable to treatment. The further statement that huskiness is more frequent in such children than in syphilitic adults, may possibly be only apparent, the cases thus reported being met with at a hospital throat department.

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[1] W. Downie, *The Lancet*, May 5, 1894.

E.—*Hereditary Syphilis in the Ætiology of General Paralysis.*

In some cases hereditary syphilis seems to issue in general paralysis of the insane. The *rôle* it may play, and its importance as a factor in the production of general paralysis, vary in different cases. It is at the basis of some of the cases of general paralysis occurring at the developmental periods of life termed puberty and adolescence. These juvenile or youthful general paralytics may present clear signs of congenital, or of other and later manifestations of hereditary syphilis; or, with a history of parental syphilis, the signs of hereditary syphilis may be indistinct or doubtful in the patients themselves.

I have briefly referred to a case, personally observed, in my work on "General Paralysis." Other cases of hereditary syphilis, apparently the chief factor, or an important factor, in the production of general paralysis at these early periods of life, have been published by Clouston (2); Ballet, A. Westphal, M. J. Nolan, Wiglesworth (probable), Middlemass (2). In Dr. Wiglesworth's[1] case, the patient's mother had had thirteen pregnancies; of these, six were "either miscarriages, still births, or died within a few weeks after birth—a history certainly suggestive of syphilis, though no other evidence of this disease was forthcoming."

Of cases reported by Dr. Middlemass, two were most probably hereditarily syphilitic; but in the others this condition was doubtful (2), or not indicated (1).

Other cases (3), certainly of hereditary syphilis, had brain disease, only partly resembling general paralysis in lesions and symptoms, as reported by Homén. It is quite doubtful, therefore, whether these three cases also should be placed or not under the above heading.

In several of these cases, a cranial injury apparently was an exciting factor, also. Such injury during the developmental epochs of life would be likely to arouse a latent inherited syphilis to overt manifestation in the damaged organ, the brain.[2] The description of a case, published[3] some years ago, of congenital syphilis and dementia in a boy aged 15, whose father died insane, is much like that of juvenile general paralysis.

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[1] *Journal of Mental Science*, July, 1893, p. 357.

[2] Injury to head, indeed, was *the only* apparent cause in a male case of general paralysis beginning at about the age of 12. Dr. H. C. Bristowe, *British Medical Journal*, Nov. 18, 1893, and May 26, 1894.

[3] Dr. Hughlings Jackson, *Journal of Mental Science*, Jan., 1875, p. 520.

F.

In a boy, born of a syphilitic mother, a spinal disease came on at the age of 6 years. He was shewn recently,[1] at the age of 32, as a case of syphilitic spinal paralysis of Erb's type. (See Part III. of this article, as to this type.)

The symptom-group of so-called *spastic spinal paralysis* may, clinically, thus be a part of a hereditary syphilitic cerebro-spinal disease; as, also, occurred in Professor J. Hoffmann's case.[2]

A boy, aged 14 years, with a history of hereditary syphilis, up to the age of 12 years was of defective bodily development and backward in mental capacity. At 12, his mental powers began to fail very much, and spinal symptoms gradually came on. At 14, there were reflex iridoplegia, mydriasis, loss of pupillary reaction in convergence, paresis of accommodation; extremely spastic gait, stiff limbs (but preserved motor power and sensory and sphincter function); mental anomalies; defective bodily growth. The condition apparently was of hereditary syphilitic origin.

The morbid changes, presumably, affected the brain cortex, the pupillary nervous mechanism, and the lateral cord-columns; these last, primarily or secondarily.

The defective bodily growth and mental backwardness, from birth, were taken to be due to an arrest or standstill in development. The recent mental changes and pupillary and spinal symptoms pointed to an active process of disease.

Besides the brain-changes, partly due to developmental stoppage and defect, it is probable that the spinal affection formed in a way the same as, or similar to, that of primary lateral-column disease of acquired syphilis,[3] or as in hereditary spastic spinal paralysis (Strümpell); or as in the spastic type of infantile on juvenile general paralysis.

PART III.

SYPHILITIC SPINAL DISEASE GENERALLY; BUT PARTICULARLY OF SPINAL CORD, WITH ESPECIAL REFERENCE TO SYPHILITIC SPINAL PARALYSIS; AND TO TABETIFORM CASES.

In 1864, Virchow mentioned syphilitic gummatous disease of spinal meninges and of spinal cord; in 1875, Wunderlich spoke of syphilis of spinal cord as being not rare. The subject was worked at by Leyden, Rumpf, Rinecker, Oppenheim, Goldflamm, Mauriac, Jürgens, and others. Gerhardt had thirty-nine cases of brain syphilis; nine of spinal cord syphilis; but, of these latter, two were cerebro-spinal. Gilbert and Lion mentioned forty-four cases of spinal cord syphilis, but twelve of these had brain syphilis also. Of Kuh's examples, many seem to be original. In Dr. Buzzard's[1] work, of thirty-seven cases, seven had spinal cord syphilis, but only one of these seems to have been a case of spinal cord syphilis only; the other six, sooner or later, had cerebro-spinal disease of syphilitic nature.

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[1] Mendel, Berlin Medical Society, November 15, 1893.

[2] *Neurologisches Centralblatt*, 1894, p. 470.

[3] As, for example, in Minkowski's case.

For the present purpose we divide the subject into:—

(1) Syphilitic disease of spinal bones, including caries and necrosis.

(2) Syphiloma of spinal meninges and of spinal cord.

(3) Syphilitic disease of larger spinal blood vessels.

(4) Syphilitic spinal meningitis, subacute or chronic (including that of the dura).

(5) Syphilitic spinal myelitis; or softening; (*a*) with spastic paralysis.

(6) Syphilitic spinal myelitis, (*b*) with taboid conditions.

(7) Tabes.

It does not seem necessary to treat of the syphilitic disease of spinal nerve roots separately. The special symptoms attending it will be referred to further on. Nor do we deal with meningo-myelitis, apart and at length.

It is proposed to take these sections in order, but to deal only very briefly with the first four; and to treat of the last, or tabes, in a separate division (Part IV.) of this communication, in some measure because its relationships are somewhat different, and partly on account of the space needed for anything like its adequate treatment.

1 or A.—*Syphilitic Disease of Spinal Vertebræ.*

Observation has led me to suppose that syphilitic disease of bone occasionally forms the predisposing basis for future tubercular disease in the (bony) part affected. But irrespectively of that, in my experience the spinal bones are sometimes affected with syphilitic disease. Gerhardt[2] also observed two such; one affecting the fourth cervical vertebra, one the eleventh dorsal, and both with spinal symptoms; and he suggested that, just as brain syphilis may descend to the cord, so may disease of the cranial bones be followed by that of the spinal; or, again, the vertebral disease may be an extension from disease of the throat. Thus, Hobbs reported a case in which ulceration had destroyed the pharyngeal wall and caries of a cervical vertebra had arisen. By the finger, a sequestrum could be felt, pressure upon which set up, transitorily, a unilateral paralysis, and, subsequently, choreic movements.

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[1] "Clinical Aspects of Syphilitic Nervous Affections."

[2] *Berliner Klinische Wochenschrift*, December 11, 1893, p. 1210.

Finally, injuries affecting the bones of the syphilitic are not to be forgotten, occasioning and localising, as they may do, not only disease of the bones, but also of the spinal meninges, apparently of syphilitic nature; and even leading to paresis, but perhaps completely disappearing under anti-syphilitic treatment.

2 or B.—*Syphiloma of Spinal Meninges and of Spinal Cord.*

The gummata may be numerous and small. We are only referring to those easily visible to the naked eye and forming small tumours. Numerous microscopical cellular collections in the walls of meningeal vessels and in those of the cord are really buds of possible future gummata; but these are not at the present moment under consideration.

More frequently, there is one gumma, and of fair size, sometimes two or more of similar size. The solitary gumma is apt to affect, more or less, one side of the cord, especially the posterior column and the lateral, and thus may give rise, more or less completely, to the symptoms of Brown-Séquard's paralysis, namely, that due to a tranverse lesion destroying, at some level, one lateral half of a tranverse section of the cord. Gummatous meningitis, or a gumma even when distinctly meningeal in origin, may, by pressure, produce the same effect as if seated in the cord. The Brown-Séquard type of spinal paralysis (namely, paralysis and hyperæsthesia on the same side, below; anæsthesia on the opposite side) may also follow a focus of softening, affecting one lateral half of the area of a transverse section of the cord, and due to the blocking of a syphilitic vessel, or of several. Moreover, that type of paralysis is the extreme example of that of which we often see minor degrees in syphilis of the spinal cord; namely, for example, a much greater degree of paraplegia in one leg than in the other, of sensory symptoms on one side than on the other, or, again, the existence of failure or perversion of one form, or of some forms, of sensation in a part, simultaneously with a normal state of other forms of sensation therein.[1]

Since this was written, the account met with of a published case, illustrates these points as to semi-lateral paralysis and sensory anomalies. Left lower limb; the paler and colder, the site of marked paresis, exaggerated tendon reflexes, hyperalgesia, and some loss of muscular sense. Right lower limb; slight paresis; up to mid-thigh, loss of sensibility to pain and temperature, that to touch normal; from mid-thigh to epigastrium, diminution of sensibility to pain and temperature.[2]

3 or C.—*Syphilitic Disease of Larger Spinal Arteries and Veins.*

As just mentioned, the syphilitic disease of blood vessels supplying the cord may lead to obstruction of the vessels, and local softening, with symptoms corresponding to the exact site of this focal alteration; as, *e.g.*, when, affecting one side of the cord, it produces, more or less fully, a palsy of the type made by experimental transverse lateral hemisection of the cord. Subsequently, the secondary degenerations, descending and ascending, consecutive to the local lesion, will modify this, and any other early clinical aspect, by superaddition of the clinical results they occasion.

This spinal arterial and venous disease is apt to accompany syphilitic meningitis, which so often is a gummy meningitis. As various cases indicate, syphilitic meningitis, and this vascular disease, are apt to be met with in, and to explain, such occurrences as local softening, hæmorrhage, sudden increase of pre-existent paralysis or paresis, or the onset of sudden paraplegia, perhaps at an early stage of the disease—perhaps even appearing almost like a precursory phenomenon.

The *arterial* disease tends to obliteration of the lumen of the vascular tube, and to local softening of that part the circulation in which is immediately cut off. The disease affecting *veins* of various sizes tends to lead to rupture and hæmorrhage.

Many cases of syphilitic paraplegia, sudden or rapid in onset, attributed to myelitis, are probably due, in reality, to the softening of portions of spinal cord, following upon occlusion of syphilitic blood vessels, and to the conditions consecutive to that softening. To this reference will again be made under section "E or 5." Such spasmodic paraplegia alleged to be from syphilitic transverse myelitis, is often, in reality, from softening of cord locally, from vascular lesion (endarteritis, &c.), on which sclerosis tends to come, in turn; or hæmorrhage into the cord may result from the vascular lesion.

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[1] Two syphilitic spinal cases with Brown-Séquard's syndrôme have since been published by Dr. J. Michell Clarke. One, at least, of these is attributed to dorsal meningo-myelitis. *Lancet*, May 26, 1894.

[2] Krafft-Ebing, *Allg. Wiener Med. Zeitung*, Nos. 5 and 6, 1894.

A case[1] appearing during revision of the MS. of this article, is one in which, preceded by some pains and rectal and vesical symptoms, flaccid paraplegia came on somewhat suddenly, as well as loss of reflexes in lower half of body. Hæmorrhage had taken place into the spinal grey matter, chiefly on the left side, affecting a vertical extent of about 1¾ inch of the mid-dorsal region of the cord. Here, and hereabouts, were also enormously dilated blood vessels; very many vessels thrombosed, some containing new-formed tissue, their walls covered with nuclei, also well-marked perivascular cell infiltration. No compound granule cells were seen, and the inflammatory changes—excess of nuclei and round cell infiltration—were just around the vessels.

The meninges also showed infiltration with round cells, and marked vascular changes (endo- and peri-arteritis, endo- and peri-phlebitis, thrombosis.)

Pathological Diagnosis.—Syphilitic disease of vessels, as above, thrombosis, hæmatomyelia, lepto-meningitis.

Clinically, the mode of onset of the symptoms pointed to a very acute myelitis rather than a primary hæmorrhage. But, microscopically, it appeared that the primary change was syphilitic disease of the vessels, followed by thrombosis, and this, subsequently, by hæmorrhage into the cord.

In a syphilitic and alcoholic case, aged 37,[2] with symptoms of locomotor ataxy, almost complete recovery from these last occurred under mercurial treatment. Eight months later, came piercing pains, some paralysis and muscular atrophy; these symptoms began in the lower limbs, later on invaded the upper limbs, and finally paralysis affected the cranial nerves; death soon following. Advanced atrophic changes were found in the motor nerves; also vascular sclerosis and foci of softening in the grey substance of the anterior spinal cornua. These latter necrobiotic changes were assigned to the chronic vascular

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[1] Dr. R. T. Williamson, *Lancet* (London), July 7, 1894.

[2] Petrini, *Mercredi Médical*, 1894.

(arteritic) lesion, and they had occasioned the fatal illness and its symptoms. The earlier and cured symptoms were assigned to a pseudo-tabes due to peripheral polyneuritis.

4 or D.—*Syphilitic Spinal Meningitis (mostly Chronic, may be Subacute).*

Syphilitic meningitis is the most common specific spinal lesion. This corresponds to two factors: (1) the elective affinity—we may use the metaphor—of syphilis for blood vessels—the pia is, largely, vessels; and (2) the great predominance of the spinal meninges. For the cord is a slender organ, and bears a smaller ratio to its encompassing meningeal muff than that of the brain to its encapsuling membranes.

Thus, the meninges are frequently attacked first, in syphilis, and when this is so, the morbid process may or may not extend from them to the cord itself.

So much, however, has been said in the first part (Part I.) of this article, when dealing with cerebral and cerebro-spinal syphilis, as to the syphilitic disease of blood vessels, and of meninges, that we need not dwell upon meningitis now. Frequently, it is distinctly of the gummy form.

Oppenheim clearly sets forth the clinical phenomena usual in syphilitic spinal meningitis:—Stiffness of the back, back pains, irradiating pain, girdle-like pain, anæsthesia, paraplegic conditions; which develop abruptly and seem to depend on one or more foci of change.

Hypertrophic cervical spinal pachy-meningitis is in some cases more or less certainly or probably syphilitic, as in two examples recently published by Köppen.[1]

Associated Disease of a Number of Spinal Nerve Roots.

This sometimes occasions certain of the most striking of the symptoms, as, *e.g.*, in cases of syphilitic spinal meningitis. Occasionally, indeed, there are examples in which multiple spinal nerve root lesion is the leading condition, as, *e.g.*, in some by Kahler and Buttersack. This predominant affection of spinal nerve roots may produce (*inter alia*) such symptoms as partial anæsthesia of the trunk, paralysis of trunk muscles which rarely, otherwise, are palsied (as, *e.g.*, muscles of respiration), also herpes zoster.

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[1] *Neurologisches Centralblatt*, 1894, p. 497.

Associated Meningitis and Myelitis.

Although, for convenience, we here have separated meningitis from myelitis, they are very often combined, and a syphilitic meningo-myelitis is a highly characteristic lesion.

Writing in 1889, on early syphilis of the spinal cord, MM. A. Gilbert and G. Lion described four types:—

Hyperæmic and necrobiotic meningo-myelitis.
Diffuse embryonic " "
Gummy " "
(Cases with gummatous nodules come here.)
Diffuse sclerotic meningo-myelitis.

Writing in 1893, they profit by the labours of Greiff, Schmauss, Moller and Sottas, to add facts relative to syphilis of the blood vessels.

In syphilis of the cord and its tunics a variety of conditions obtains; of these the diffuse infiltration of the meninges affects the whole structure of the pia, with the walls of the vessels; whereas, in the arteritis existing alone, the arterial coats, only, are affected. The conditions may be grouped as:—

(*a*) Phlebitis and arteritis.
(*b*) Diffuse embryonic infiltration of meninges; and of their prolongations into the cord-substance.
(*c*) Gumma.
(*d*) Dilatation (ectasy) and multiplication of capillaries.
(*e*) (1) Softening, and (2) Sclerosis.

Some of these are primary or initial; others are secondary. For "(*a*)" "(*b*)" and "(*c*)" are primary or initial; and probably "(*d*)" (?) is so also. And of "(*e*)" the constituent factors (*i.e.*, softening and sclerosis) are secondary. "Still, it is not sure that sclerosis may not sometimes be initial."[1]

Yet, after describing a case of hereditary syphilitic cerebrospinal disease, in which (*inter alia*) was chronic gummatous syphilitic meningo-myelitis, invading posterior, and postero-lateral columns and grey matter, Böttiger[2] holds that the lesions, as also acute myelitis following lues and the chronic meningitis in tabes, are not at all characteristic of syphilis.

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[1] Gilbert and Lion. *Comptes Rendus de la Soc. de Biol.*, Séance, April 22, 1893.
[2] *Neurologisches Centralblatt*, 1894, p. 497.

5 or E.—*Syphilitic Myelitis, mostly Chronic or Subacute; or Softening.—Syphilitic Spinal Paralysis.*

Forms of syphilitic change of the anterior cornual grey may occasion muscular atrophy, or acute ascending paralysis (of Landry). But these are not taken up here.

The recent attempt of Erb to establish a clinical form of syphilitic disease, which is to be taken as syphilitic spinal paralysis, *par excellence*, has proved to be the trumpet call to a lively fray.

A number of observers had previously described the clinical or pathological aspects of, or of supposed, syphilitic spinal myelitis, such as Erb himself,[1] James Ross, Breteau, W. H. Hammond, Caizergue, Julliard, Proux, Vinache, Fournier, Oppenheim, Eisenlohr, Sachs. Most of these mentioned facts or clinical features correspondent to some or others of those upon which Erb relies as, in their association, establishing the syphilitic spinal paralysis, he describes, as being a clear and distinct form of disease. Suffice it to mention the prior observations of a few upon this subject.

Thus, Oppenheim[2] had declared that syphilis of the spinal cord often enough had the clinical form of acute and subacute myelitis, if only one stage of the disease was kept in view, and the preceding clinical history, and course, disregarded. For differential diagnosis, he relied on the simultaneous or preceding brain symptoms, the paroxysmal or stroke-like course of the disease, and the fluctuation of individual symptoms; the symptoms referable to meningitic irritation and to the nerve roots; those denoting a multiplicity of foci; or, in an incomplete form, those due to transverse lesion of one lateral half of the cord. These views were more or less confirmed by Siemerling, Marinesco, Pick and others.

Eisenlohr,[3] also, had stated that spinal syphilitic disease, on the clinical and symptomatological side, for the most part presented symptoms which correspond to a subacute or acute transverse myelitis (paraplegic symptoms, &c).

Sachs[4] had stated that this syphilitic invasion "would seem to attack the lateral columns most frequently; the posterior columns come next in order, and the grey matter last of all." "Starting from the meninges, the process may invade the substance of the cord or the brain; in the majority of cases the lateral columns will be the first to be attacked and spastic paralytic symptoms are the result."

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[1] "Krankheiten des Ruckenmarks," *Zweite Auflage*, 1878.

[2] *Berliner Klinische Wochenschift*, 1889, Nos. 48 and 49.

[3] "Separat-Abdruck a. d. Festschrift zur Eröffnung des Neuen Allg. Krankenhauses zu Hamburg-Eppendorf," p. 3, 1889.

New York Medical Journal, September 19, 1891.

Joffroy[1] had described a case of spinal cord syphilis which presented the contrast between great increase of tendon reflexes and slight muscular rigidity or contracture, on which Erb subsequently laid stress.

Th. Rumpf[2] had published a case with similar contrast strongly marked; and another,[3] due to specific disease of blood vessels of spinal cord, which presented nearly every point detailed by Erb.

M'Call Anderson[4] had described two cases which, practically, presented Erb's symptom-group, with the exception that, inasmuch as the rigidity, spasm or cramp of the lower limbs were highly marked, as well as the exaggeration of tendon reflexes, the contrast just mentioned was absent, in them.

It will be seen, presently, how these prior descriptions refer to cases of the type of those from which Erb drew the symptom-group which he put forward as that of a distinct, separate, clinical form of disease ; but the pathological anatomy of which he left for the future, mainly, to reveal with precision.

To plunge at once into the immediate subject ; Prof. Erb, of Heidelberg, recently,[5] and his assistant, Dr. Sidney Kuh, subsequently,[6] and more fully, described a symptom-group under the name of "syphilitic spinal paralysis"—a type of spinal cord disease which manifests itself in, and is characterised by, a spastic paralysis of the lower limbs, highly increased tendon reflexes, slight muscular rigidities, disorders of urinary evacuation, proportionally slight sensory affection. There are other less important features ; and expressed a little more in detail. This symptom-group, which, at a superficial glance, presents the aspect of spastic spinal paresis or paralysis, comprises, clinically, on analysis of the description given, the following, namely :—

(1) The features of spastic spinal paraparesis, or even para-

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[1] *Gazette des Hôpitaux*, No. 116, 1882.

[2] "Ueber die syphilitischen Erkrankungen des Nervensystems," Wiesbaden, 1887, p. 328.

[3] *Op. cit.*, p. 351.

[4] "On Syphilitic Affections of the Nervous System," 1889, pp. 47 and 49.

[5] *Neurologisches Centralblatt*, March 15, 1892.

[6] *Deutsche Zeitschrift fur Nervenheilkunde*, Band iii., Heft 6.

plegia, often not equally affecting the two limbs, manifested in attitude, movements, and in gait, which is usually of the stiff-legged, spastic type, and contrasting with the relatively much slighter paresis.

(2). Great increase of the tendon reflexes.

(3) Muscular rigidity or contracture only slight, if compared with the greatly enhanced deep reflexes.

(4) Weakness and functional disorder of urinary bladder and rectum.

(5) Sexual failure, or impotence.

(6) Comparatively slight, but distinct, sensory symptoms; severe pain unusual.

(7) The onset ordinarily gradual; the development, occupying weeks, months, and often years, is occasionally more rapid.

(8) Improvement is frequent.

These are the peculiarities and conditions special to the affection in question.

As to the eighth and last; according to Erb an important peculiarity is that the paraplegia may revert to paresis, or even apparent recovery may take place, and walking may be resumed, but, if so, the tendency is to relapse, the paresis, &c., recurring.

Between the cases occur the most manifold differences in intensity of the different symptoms. The above is only the type. The condition may become stationary for years together.

The symptoms are attributed to a partial transverse lesion of the dorsal portion of the spinal cord: a local myelitis.

Having with these few strokes limned the condition in outline, the importance of the description, and the desirability of making it full and clear in order to compare with it the experience of others, necessitate a more detailed account of the several clinical conditions mentioned, and we speak of the clinical manifestations, firstly, at the commencement of the disorder, and secondly, when it is fully developed. Kuh's account is followed here.

I.

I.—*At the commencement and early stage* of the condition, the initial phenomena of the spinal disorder creep on in persons who a few years previously had primary or secondary syphilis. Some of these have previously had indications of brain syphilis, such as diplopia, optic neuritis and visual failure; or even more severe ones. Usually, there are slowly increasing weakness and stiffness of lower limbs, often with paræsthesiæ, mostly as formication in the legs, but in one case affecting the epigastrium. In some cases are girdle sensation, or sensation of burning, of electricity, of shivering in legs, of dragging from back to legs, or sensory perversions about the anus. Hyperæsthesia is occasionally an initial symptom. Pains proper, if present, are almost always slight, and most frequently are felt at the vertebral column or sacral region; sometimes they take the girdle form; they may be felt in different parts of the trunk and lower limbs. Spasmodic movements in the legs may occur. Anæsthesia is rare.

The above sensory and motor disorders not seldom affect one leg first, and the other only later on. The spastic paresis may be more or less limited to one lower limb only. More seldom is there an ascending invasion of symptoms, gradually extending upwards from the feet and halting only high up.

Important at this period, and sometimes long preceding the others (in one case by three years), are the disorders of urinary evacuation; in over one-half of the cases were they early symptoms. Most frequently there is more or less paralysis of the detrusor urinæ. In other examples is weakness of the sphincter; in one case both retention and incontinence. In the majority of the cases with these urinary symptoms is also disorder of fæcal evacuation, having the form of constipation more often than looseness.

Not seldom an early symptom is weakness of sexual power, which tends, anon, to attain impotency. But the sexual phenomena may be those of excitation, *e.g.*, priapism; in one case severe back-pain, with sexual excitement, which at times increased to seminal ejaculation.

Precocious severe paralysis is rare. Complete paraplegia of acute onset is observed only in rare, and otherwise exceptional, cases.

II.

II.—*When the condition is fully developed* the clinical features include in the first line a spastic paralysis of lower limbs. It is met with in every degree, but the lighter grades of it predominate. Very seldom is the paralysis complete, and, if becoming complete, it is only temporarily so. To this rule there are only few exceptions, and these concern fatal cases, only coming under observation late on in the affection, or during a relapse. The ordinary state is a very spastic gait, with relatively slight paresis, and the tendency for existing paralysis to revert to paresis. Even in this stage are the motor disorders and failure often more marked in one limb than in its fellow—sometimes even much more marked, in the earlier parts of this stage.

Should there be certain complications, other motor symptoms occur, but do not require mention here.

In contrast with the extremely spastic gait is the slight muscular rigidity or contracture. Erb found this contrast in nine-tenths of his cases. Noted in a few cases by others previously, it appears, for the most part, to have escaped notice or mention. A high degree of muscular rigidity and contracture, if present, occurs almost exclusively only in far-advanced cases.

The tendon reflexes of the lower limbs are always enhanced; ankle clonus is almost always found, and patella clonus very frequently. Only seldom are the deep reflexes increased or affected in the upper limbs.

The skin reflexes seem to be altered in only a few cases, and usually diminished. This harmonizes with the slightness of the sensory disorders.

Sensory disorders, indeed, are almost never entirely absent, but are very frequently restricted to subjective disorders, or to slight, incomplete or quite circumscribed decrease of a sensation. By the sensory alteration one lower limb is sometimes more strongly affected than the other; but still more often are there only small, circumscribed parts affected, and especially so as regards the objectively demonstrable disorders. Thus, there may be isolated diminution of the tactile-, pain-, muscular-, or space-senses; staggering on eye-closure; associated lessening of sensibility to pain and temperature, or to pain and touch, or to temperature alone.

Extensive anæsthesia is quite exceptional only; and, if present, only temporarily so, or at the final stage.

Hyperæsthesia sometimes affects leg, thigh, or back, and in isolated cases may be limited to the sense of cold, or of cold and heat, or of touch.

Paræsthesiæ are seldom absent. Such are formication, numbness, feeling of tension, of burning, &c.

Occasionally is the girdle sensation in lower limbs or trunk. Severe pains occur very seldom. Slighter pains are tolerably frequent, and may be girdling pains, irradiating pain in legs, or lumbar or dorsal pains, or confined to single nerves.

Spasm may affect the leg or calf.

Symptoms relating to sexual function and the urinary bladder continue over from the early stage.

Bed sores and cystitis are infrequent, and, if present, appear mostly in very severe cases only, or in the later stages.

Syphilis of skin, or of mucosæ, may be present, or the scars it has left behind.

In pure uncomplicated cases the symptoms are restricted to the lower half of the body; and the above accounts exhaust them.

The differential diagnosis must distinguish tabes, insular sclerosis, compression myelitis, syringomyelia, primary spastic spinal paralysis, hæmatomyelia, ataxic paraplegia.

Besides these, which usually offer no extreme or very special difficulties, is *non-syphilitic transverse dorsal myelitis.* Cases of this latter form are distinguished by—the presence of atrophy, by muscular rigidity, or contracture proportional to the spastic paresis with its heightened tendon reflexes, by more persistent paraplegia, by more lasting diffuse sensory disorders implicating more kinds of feelings equably, by the absence in some cases, the slightness or lateness in others, of disorder of urinary and fæcal evacuation.

Very rare in non-syphilitic cases, very frequent in syphilitic, is the symptom-group :—Spastic paresis of lower limbs with increased tendon-reflexes ; very slight muscular rigidity or contracture ; more marked implication of bladder or rectum ; slight (or even absent) disorders of sensibility, local, and limited to one, or some only, of the modes of feeling.

Pathological Anatomy (inferential).

The lesion is somewhere in (the lower half of) the dorsal cord, as determined by the height to which the sensory alteration usually attains ; by the usual exemption of the upper limbs ; and by the absence of loss of knee-jerk.

The transverse section of cord is only partly lesed ; apparently the posterior parts of lateral columns are chiefly affected, the posterior columns less affected (the posterior root-zone of which is only slightly affected as a rule), the posterior horns are also lesed (vesical, rectal and sensory defects).

(Instead of myelitis, may not the change, in some cases, be due to syphilitic spinal arterial alteration, leading to gradual occlusion, and this to slow ischæmia and softening of spinal cord locally, followed by regressive changes in the softened patch, and by systematic secondary degenerations, as well as leading to increase locally of capillary circulation and of interstitial tissues?)

In reference to the above description of Erb's alleged type of syphilitic spinal paralysis, have already been mentioned earlier descriptions or statements than his, evidently relating to cases of the same kind.

The questions at issue are whether what Erb describes is *the* syphilitic spinal paralysis *par excellence*, as he seems to think; or whether it represents merely one of several or many such forms; or whether it a only a phase in what ought to be a wider clinical conception and statement; and whether the forms are not in reality protean in their aspects, because so mixed and varied in site of lesions and in symptoms as that a distinct clinical entity cannot be established.

The view of the present writer, as to this form of syphilitic spinal paralysis described by Erb, to which the latter gives extraordinary significance as a separate, special, distinct and chief form of syphilitic spinal, paralytic disease, may be briefly stated thus:—

Syphilis usually attacks several parts of the cord, and its appendages, or anatomical out and ingoings, and thus usually does not confine its ravages to one column or system of fibres, but expends them on several.

This being the case, I do not find anything very especial or peculiar in the fact of the predominance of the spastic type of the spinal paralyses produced by syphilis. For example, in ordinary ataxic paraplegia, in which the chief lesions are changes affecting the posterior and part of the lateral columns of the cord, the mixed and modified symptoms of affection, both of the posterior columns and of the crossed pyramidal tracts, gradually undergo transformation in the later stages, so that the clinical garb of a spastic paraplegia is put on. Seen now for the first time, and without accurate knowledge of the preceding clinical history, the case may possibly be taken as one, throughout, of the spastic type.

In Erb's type, the symptoms distinctly point to parts of the cord other than the pyramidal parts of the lateral columns being also lesed, although in less degree than it. Even this comparative diffusion of the change does not, however, cut the syphilitic cases off sharply from other cases, or from examples of somewhat similar cognate disease. Thus, in primary spastic spinal paralysis, attributed to lesion of the crossed pyramidal tracts, there is in some cases also degeneration of the postero-median and postero-external columns, and of the direct cerebellar tract; instead of merely an isolated systematic lesion in the first-named tracts.[1]

In other, and non-syphilitic, paralyses, also, paraplegia may meliorate into paraparesis.

In short, I think we may regard spastic spinal paralysis as being not merely *the* chief syphilitic spinal paralysis, *apparently* as regards *frequency;* but that this conception is to be merged into a wider one, applying to most cases, whether syphilitic or not, namely, that clinically, spastic spinal paralysis in its different varieties is *the columnar* spinal paralysis *par excellence;* not the only one, but the one to which not only those primarily spastic belong, but to which, also, several forms tend which are not originally of that aspect, or are only partly so.

Apparently as regards *frequency,* I have expressed it just above; for in many cases the spastic spinal paralysis symptom-group only forms one phase or stage of a long and variegated course of a syphilitic spinal malady.

This view leaves due room for the atrophic and the flaccid spinal paralyses, and lesions predominantly of the grey, whether of syphilitic nature or not.

We now turn to some cases reported since Erb's article appeared, and to some contributions made to the study of this point.

Dr. B. Sachs[2] describes four cases bearing on this subject, but none of them elucidated by necropsy.

In the first case was complete spastic paraplegia, with greatly exaggerated tendon reflexes; complete paralysis of upper limbs without increased reflexes, but with some atrophy of interossei. When first seen, no sensory symptoms; subsequently, severe anæsthesiæ in lower limbs and lower trunk; highly marked muscular contractures in lower limbs; bedsores; cystitis.

In the second case, extreme and absolute spastic paraplegia; extreme contractures; complete anæsthesia, passing up to two inches above the symphysis pubis; no vesical, or rectal, or trophic trouble.

In the third case, extreme spastic symptoms, with contractures, slight bladder implication, no sensory or atrophic symptoms.

In the fourth case, paralysis and more or less atrophy of all limbs; one knee-jerk feeble, the other lost; all superficial reflexes lost, no vesical or rectal change, and no bedsore; large area of anæsthesia on the trunk (slight cerebral involvement, ocular symptoms).

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[1] *E g.*, cases by Dr. Ad. Schule. *Deutsche Zeitschrift für Nervenheilkunde*, Bd. iv., p. 161.

[2] New York Neurological Society, April 1893, BRAIN, 1893, p. 405.

Thus we see how each of these deviates from Erb's type; how in one or more of the four cases there are points of opposition to, and difference from, that type, as regards each of the clinical features which constitute it a typical symptom-group; unless it be the last one, namely, the tendency to improvement. Contrary to the type, in one there was widespread atrophic paralysis, and the deep reflexes were lessened or lost;—in three of them there was spastic paraplegia of most complete degree—a deviation from the type—and in these three the tendon reflexes were greatly exaggerated. In one or two of them the muscular contractures were slight; in two of them they were extreme—thus differing from Erb's type. In one case there was permanent implication of the bladder, in the others no involvement—a departure from the alleged type. In all but one of them sensation was disturbed, but in some there was extreme anæsthesia—a deviation as to type. In two the onset was gradual; in two quite sudden, the latter unlike the type. In all, was decided tendency to improvement.

Sachs looks upon the points more characteristic of spinal cord syphilis than are any other, as being:—

(1) The first and most striking one—the unusual distribution of the disease over the greater portion of the cord.

(2) The relatively slight intensity of the morbid process, and consequently of all symptoms, as compared with the extensive area involved, as evidenced by the preservation of some of the functions of the cord with complete loss of others.

(3) A rapid dwindling of some of the symptoms, and the very chronic persistence of others. In some, *e.g.*, the anæsthesia lasted but a very short time, whereas the paralysis was recovered from very slowly.

(4) The very frequent history of other symptoms pointing to specific disease in the same or other parts of the central nervous system.

[(5) The tendency to remissions and relapses.]

Of the above, I do not grasp the significance of the "evidence" adduced for No. 2.

In discussion, Dr. E. C. Seguin stated that the irregularity or lack of completeness of the symptom-group is very characteristic of these cases of spinal cord syphilis; a view in which Dr. Fry coincided, inasmuch as some of them will strongly simulate tabes, others ataxic paraplegia, others ordinary transverse myelitis; yet with an absence of important symptoms, which not only excludes them from these groups, but often, as well, raises the suspicion of syphilis.

Dr. H. Lamy[1] also reports cases clinically more or less of Erb's type. Some are typical instances, others represent it fairly well. In two other examples, Erb's syndrôme was accompanied with reflex light, and accommodatory, disorders of pupils, or palsy of eye muscles.

After a premonitory stage, comes syphilitic paraplegia, which may be of ordinary or of severe and acute kind. In an example of this latter kind, however, Nonne, of Hamburg, found only the signs of an acute myelitis of idiopathic type, and no specific spinal changes whatsoever.

Diffuse meningo-myelitis being the most frequent form of syphilitic spinal cord disease—although lepto-meningitis or pachymeningitis may be the form taken—the specific disease of the blood vessels accompanying it begins in the meninges; of the several kinds of blood vessels, is earliest and most in the veins, and in them, usually begins in the adventitia. The spinal moningitis is often accompanied by basilar cerebral meningitis.

In the prodromic period this syphilitic condition preferably takes its expression in headache, vomiting, vertigo, transitory comatose state, or cranial, especially ocular, nerve palsy; or backache of specific character, from meningitis.

In the second period (usually) is spinal paralysis, mostly as a moderately severe transverse dorsal myelitis (spastic paresis of lower limbs, slight disorder of sensation and of sphincters), less often as severe acute palsy, perhaps from arterial thrombosis, or from acute central myelitis, which usually is promptly fatal.

Déjérine[2] holds strongly that in common syphilitic paraplegia of sudden onset the spinal lesions are not myelitic, but consist of softening, from endarteritis, with subsequent sclerosis.

As well as in acute sudden syphilitic paraplegia, so also in fairly chronic spasmodic syphilitic paraplegia, the pathological anatomy may be that, not of myelitis, but of local softening and degeneration of the cord.

Syphilitic inflammatory changes of walls of veins and arteries locally, and perhaps the formation of miliary pia-matral gummata, constitute the first period, or that of meningo-vascular alteration, as in cases by M. J. Sottas.[1] If these lead to arterial obliteration, a period of local softening follows; to this succeeds a period of degeneration of nerve fibres and nerve cells at the site of lesion; and, subsequently, consecutive degenerations, whether ascending or descending. Then supervenes a period which he terms that of inflammatory reaction; but in which the vasa-vasorum of the obliterated vessels are dilated, show in great numbers (capillary new-formation?), and therewith is increase of interstitial elements (period of sclerosis).

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[1] *Nouv. Iconogr. de la Salpêtrière,* 1893, No. 4; and "De la Méningo-Myélite Syphilitique," Paris, 1893

[2] *Comptes Rendus Hebdom. des Séances de la Société de Biologie, Séance du* 22 Avr., 1893.

Often, it is particularly the smaller and smallest terminal blood vessels of the cord; or the vascular and lymphatic systems of the pia, the nourishing membrane of the cord; which manifest the syphilitic inflammatory change. As a result of this there may be softening or death of this or that part of the cord—sometimes the marginal zone, or periphery; the grey or postero-lateral column; sometimes a complete transverse segment of the cord. Infiltration of the nervous substance itself may assist; but the changes are mainly due to the ischæmia and circulatory disorders attributable to the meningeal and vascular syphilitic lesions; and may be gradual, or, on the other hand, may be rapid or sudden.

Professor H. Oppenheim[2] believes that Erb's attempt to nosographically delimit the symptom-group he describes, and to erect it into a disease, is fraught with great difficulties, and that the enlightenment he hopes to get from the morbid anatomy in the future will turn out in a sense other than he expects.

He thinks we must take into our estimate the fact that by far the most frequent form of myelitis is *dorsal* myelitis, and this partly because of the much greater length of the dorsal cord, as compared with the cervical or lumbar portions of it, and therefore its greater liability to compression, and to invasion, by syphilomatous outgrowths, or syphilitic morbid processes.

But dorsal myelitis is evidenced by spastic paresis of lower limbs, with bladder troubles and sensory disorders. What Erb describes is the same exhibited by patients in whom the transverse lesion is incomplete or partial.

Oppenheim thinks the true prototype—the chief form—of spinal lues is universal meningo-myelitis, a diffuse inflammation and new formation proceeding in, and from, the spinal meninges, tending to invade the nerve roots and the cord; or, by forming outgrowths or tumours, to penetrate the latter.

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[1] *Comptes Rendus, Société de Biologie*, Séance, 15 Avr., 1893.

[2] *Berliner Klinische Wochenschrift*, August 28, 1893, p. 837.

Usually, the vascular apparatus participates in the disease, and often the same, or some allied, specific intracranial disease occurs simultaneously.

He reminds us that Erb guards himself against the identity of the type he describes, with syphilitic meningitis or cerebrospinal syphilis, when he adds that these may be combined with it, and that the distinction between them presents no difficulties.

To arrive at a final tenable conclusion, the future must reveal more of the morbid anatomy; and, for the time being, he would refer Erb's type to the known forms of spinal syphilis, particularly syphilitic meningo-myelitis, in the course of which it forms a stage, and represents the type of the cases of this kind possessing a relatively more favourable nature, and of chiefly dorsal spinal site. But it is *not* a disease *sui generis*.

As already stated, owing to its length, the dorsal part of pia and cord is that most likely to be affected. Early treatment, in the better classes of society, leads to improvement or temporary recovery, the meningeal process recedes, the specifically syphilitic morbid processes in the cord make no advance, the sequelæ alone remain; namely, myelitis, atrophy, secondary degeneration; and as a permanent condition, with its symptoms, usually remains partial dorsal myelitis; in other words, "the syphilitic spinal paralysis," so-called, of Erb's type. Erb's symptom-group holds as a nucleus in very many such cases, but in most only forms one epoch in the course of the disease. Necropsy only comes, as a rule, when the disease is far advanced, or has spread to the medulla oblongata, or to the brain; and whilst often the morbid process has led to incomplete, sometimes focal, dorsal myelitis with secondary degeneration of pyramidal tracts, the morbid changes are almost never limited to these. So he maintains.

Therefore, it may be conjectured that the type described by Erb is not a disease but a stage, a group of phenomena conditionated by the particular localisation of an already known morbid process (Oppenheim).

The cases on which that type was founded were in the stage in which they could still travel and consult a specialist such as Erb. Hence a preponderance of such cases in his practice, and a seeming, but only specious, conformity of very many of them to a particular clinical type.

Not that Oppenheim, who opposes Erb's type, deems him to have judged only by the condition present at the time of examination. He does not suggest that. "But how much of the history, development and course of the case remains concealed from us when we undertake the examination at a given stage."

Considering the entire course, the clinical form of spinal lues corresponds only in a minority of cases to Erb's spinal paralysis. And other parts of the cord, or the nerve roots, or brain, or medulla oblongata, or cranial nerves, often participate; so that there are all possible transitions and blendings, from simple spinal paralysis to typical multiple cerebro-spinal syphilis.

Nor is Erb's type actually characteristic of syphilis. Any other diffuse disease of the dorsal portion of the cord, particularly any incomplete transverse myelitis, may lead to the same clinical condition; and even a similar course, as, *e.g.*, the return of paraplegia to a milder, merely paretic, condition may occur in other forms of myelitis.

Oppenheim asserted that the only characteristic points are the paroxysmal course in bouts, the effects of specific treatment, and the diagnostic criteria which I have already quoted from him above, at the beginning of this section "(5)" of this "Part III."

Professor Gerhardt,[1] however, adheres to Erb's views on this subject, and alleges that the majority of the cases at his clinic represent Erb's type; spastic paraplegia, gradually developed; exaggerated tendon reflexes; relatively insignificant muscular contracture.

The image set up by Erb he holds to be a signalising, easily recognisable one, and in the cases he observed there were acute forms of disease, with other progressive phenomena, gradually developed, and representing the disease proper, not residua. We have already seen that, on the contrary, Oppenheim regards them as being residual. Morbid anatomy must clear up the point.

Meanwhile, provisionally, he thinks a particular place must be assigned to this easily recognisable and frequent form of spinal syphilis. In his cases, the average interval from date of syphilitic infection was four years, the prognosis was comparatively favourable, rapid advance or danger infrequent. This form of disease, in which improvement or even an approach to recovery is frequent, which, according to this view, be it right or wrong, exists by itself, and proceeds from no other, may be accepted as having syphilitic disease, sometimes, as cause.

Since this article was written, a case presenting Erb's clinical type, and another which perhaps had been of that type, but not seen until years after the establishment of a considerable amount of recovery, have been published by Dr. Aldren Turner.[2] One case, aged 20, of syphilitic meningo-myelitis, by Dr. Michell Clarke,[1] differs much from Erb's type; but another case of his, aged 39, resembles that type pretty closely. Feilchenfeld's[2] case differs from it very decidedly.

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[1] *Op. cit.*

[2] *The Lancet* (London), May 5, 1894, p. 1124.

Of three cases of syphilitic spinal cord disease observed by Dr. F. R. Fry,[3] only one was spastic. They were reported chiefly for the sensory symptoms.

In the first case were sudden darting pain in lower limbs, absent knee-jerks, plantar reflex sluggish, slight Romberg's symptom, no ocular symptom. In all parts below the upper third of thighs was fair or good tactile sensibility, that to temperature and pain being lost. Later on, this sensory condition rose higher. No paresis; slight hesitation at commencement of urination. A weight crushed a great toe painlessly. Later on, involuntary micturition, rapidly failing sexual power, "vomiting and coughing spells," right lumbar pains.

In the second case, knee-jerks absent, plantar reflex exaggerated, no (relevant) ocular symptoms; tactile sense normal, pain sense totally lost from lower thirds of thighs downwards, impaired thence upwards "to waist-line," and in the same parts temperature sense much disordered and delayed, but not entirely lost anywhere. Symmetrical paresis of lower limbs, and sharp darting pains in them. Inco-ordination: Romberg's symptom.

In the third case were spastic gait, markedly exaggerated knee-jerks and plantar reflexes, slight right ankle clonus, slight Romberg's symptom, lower limbs felt weak and shaky, and ached, temperature and pain senses lessened or lost in all parts from navel downwards, tactile sensibility preserved in the same regions.

These cases are mentioned here in relation to the peculiar sensory symptoms by which they join—on the one hand, with some of the phenomena of cases of Erb's type (see above) in which were the combined loss of pain and temperature senses; or of pain and tactile senses, or loss of temperature sense alone, other forms of sensibility in the same parts being intact—and on the other hand, with syringomyelia of lower half of cord; although no mention is made by Fry or Kuh as to any likeness to syringomyelia in relation to their cases. Yet these cases—and those of cervical tabes—would form an interesting study in comparison with forms of syringomyelia. Sachs, indeed, has recorded a case in which the differential diagnosis was not determined. In the second and third sections of this Part III., cases have been mentioned partially resembling syringomyelia; also examples of Brown-Séquard's syndrôme.

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[1] *The Lancet* (London), May 26, 1894, p. 1299.

[2] Berliner Med. Gesellsch., Nov., 1893.

[3] *Alienist and Neurologist*, July, 1893, p. 389. "The Sensory Symptoms in Three Cases of Syphilitic Spinal Cord Disease."

Dr. C. E. Beevor[1] has described an interesting case of syphilitic tumours of the spinal cord with symptoms simulating syringomyelia. A gardener, aged 50, after exposure to wet, had, rapidly in succession, weakness of left leg, numbness of right knee, weakness of left arm. When admitted, two months later, there were wasting of both upper limbs, especially the left, and weakness of left lower limb. Sensation of pain, heat and cold was lost in right leg and right half of trunk up to the fourth rib; the analgesia subsequently extending to parts of forearms. Tactile sensibility was normal. Two syphilitic tumours were found on either side of the brachial enlargement of the cord—the cord was soft—apparently their pressure from without on the cord had produced the symptoms, as of syringomyelia.

In the first two of the cases (Fry's) mentioned above, the posterior columns and root-zones, and in all three the grey matter would appear to have suffered. These two cases lead naturally to those next to be mentioned, as constituting the ensuing section "(6)" of this part, conjointly with which, indeed, they form the connecting link with, and transition to, the Fourth department of our subject, namely, the relations between syphilis and tabes. For our next work is to briefly mention, with their bearing, the cases of Eisenlohr and of others.

6 or F.—*Syphilitic Myelitis, Chronic or Subacute, with Taboid Manifestations.*

Dr. C. Eisenlohr[2] recalls to mind that as to the question of the ætiological connections of syphilis and tabes, neither has an affinity of the posterior columnar disease (tabic) in histological character, to syphilitic changes, been shown, nor a constant sign of that disease capable of establishing its syphilitic nature, nor anatomical distinctions between syphilitic and non-syphilitic tabes. And that, if syphilitic disease of the vessels be taken (Rumpf) as being productive of secondary and pathological changes of tabes, then are we met by the observation (Krauss), that vessel alterations often found in tabes are alike, whether syphilis has preceded or not.

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[1] Clinical Society, London, *Brit. Med. Journ.*, Nov. 18, 1893.

[2] "Festschrift zur Eröffnung des Neuen Allg. Krankenhauses zu Hamburg-Eppendorf," 1889.

Also by Greiff and Julliard it was affirmed that the proper syphilitic disease of the spinal cord consisted of inflammatory processes in meninges, of exudative process in blood vessels and their sheaths, of hyperplasia of neuroglia, and of the effects of these upon the nervous elements; and Jürgens concluded that in syphilis the degenerations of the spinal cord and nerve roots are always deuteropathic processes, or secondary degenerations in the neuropathic sense, and that system diseases of the spinal cord are referable to other morbid states, and never, or extremely seldom, stand in any genetic relationship with syphilitic disease of the cord and its membranes.

Referring to the questions previously raised, whether there is a form of syphilitic disease of posterior spinal columns, not anatomically of the typical tabes type, but consisting of specific syphilitic changes with non-typical localisation in the spinal cord substance, and whether these, pardonably so-called, "anomalous cases of tabes" may really be divided off from classical tabes, Eisenlohr brought forward two cases as bearing on the subject. They were, in reality, examples of cerebro-spinal syphilis, yet more important (to us here) from the spinal than from the cerebral side. Both brain and cord were affected; in the first case probably simultaneously, while in the second it is likely that the cord suffered first. In both cases the posterior columns suffered most, but the localisation of the degeneration and other changes differed from that of typical tabes.

In the first case were spinal chronic diffuse meningitis (all membranes) of fibrous and of gummatous (gummy tumour) nature, and chronic diffuse interstitial myelitis.

In the second case were spinal fibrous meningitis (all membranes), and syphilitic chronic diffuse fibrous myelitis. The sensory disorders, the ataxy, &c., were of the type of tabes, and the root-zones were lesed.

As differences between the typical localisation of cervical tabes, and the ubity of the posterior columnar degeneration in his second case, he relies on the implication, in his case, of the posterior external fields of Strümpell, a somewhat different configuration of the affected area in the "cuneiform" columns, and the much sharper delimitation of sound and diseased areas.

Yet both cases presented close similarities to tabes, and were taken as examples of "anomalous tabes." Tabes may be complicated with cerebral paralysis of progressive type, or may be complicated with paraplegia, as his first case was. Peripheral nerve change may have co-existed.

In both cases were spinal cord diseases issuing from a specific chronic meningitis, and quite predominantly localised in the posterior columns, symptoms of which, in the later periods of observation, were closely assimilated to tabes. He holds that, whilst not affecting the question of the syphilitic nature of tabes, they well illustrate the fact that forms of disease dependent upon syphilis occur, clinically very like tabes, the anatomical conditions of which are quite different, notwithstanding a similar localisation. The special localisation of the process of disease conditionates the clinical analogy.

Fournier and others hold that no characteristic differences exist between syphilitic and non-syphilitic tabes; therefore, small deviations from the typical tabes must be carefully weighed, and the true nature of examples of anomalous tabes examined. So-called "syphilitic tabes" probably not seldom includes cases referable to conditions deviating from those of "typical tabes."

Cases more or less of similar import to those of Eisenlohr have also been published by others,[1] mostly cases of specific meningitis, invading the posterior columns and horns, simulating and diagnosed during life as tabes or as anomalous tabes, and some of them presenting more or less typical posterior sclerosis.

In one case were many recurring transitory nervous and mental symptoms, including pareses of various cranial nerves, and disorders of speech and of gait; finally, paralysis of left oculo-motor nerve, with reflex iridoplegia; atrophic paralysis in distribution of the right ulnar and median nerves. *Necropsy.*—Partial degeneration in Burdach's columns in the cervical enlargement, and slightly in Goll's columns in lower cervical and two upper dorsal segments. Lesions of anterior grey horns and cervical roots. Bulbar lesions. Partial lesions of brachial plexus, especially in ulnar nerve; gummatous cerebro-spinal endo-arteritis. Syphilis of ten years' standing.[2]

The case described by Dr. J. Hoffman[3] (*loc. cit.*) was that of a male, aged at death 36. At 20 he acquired a syphilitic ulcer. No secondary symptoms. Treatment by inunction.

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[1] Ewald, cited by Gerhardt, *loc. cit.*; Kuh, *Archiv. für Psychiatrie*, Band xxii.; Dinkler, *Neurologisches Centralblatt*, Band xi.

[2] *Deutsche Zeitschrift f. Nervenheilk.*, t. iv., 1893. By Dinkler.

[3] J. Hoffman, *Verhandlungen des Naturhist. Med. Vereins zu Heidelberg*, N. F., Bd. iv., Heft 4, 1890.

Chills and mental strain were alleged factors. Clinically, the case was a typical tabes, with tabetic joint disease. Mind, speech, and eye muscles normal.

Necropsy.—Degeneration in posterior columns, posterior horns and posterior nerve roots, as in "classical tabes dorsalis." Tabetic disease of both knee joints. Hyperostosis of cranium.

Microscopically. — Besides the degeneration of posterior columns, horns, and nerve roots, in the form characteristic of pure tabes, was a moderate patchy spinal lepto-meningitis, not only over its posterior surface, but, in the cervical region, equally affecting the anterior surface. Furthermore, there was a similar meningitis over the whole cerebral cortex, especially the left insula. At the brain-base the changes were insignificant.

Thickening of the vessel walls, particularly the arteries, with narrowing of the lumen; accumulations of round cells in the vessel wall, sometimes only affecting a short tract of it; extravasation of hæmacytes; formation of miliary gummata; in places quasi-hyaline-degenerated masses.

Left insula; its cortex infiltrated with cells, especially collected around the vessels, which, in unusual numbers, penetrate the cortex from the thickened pia. Cauda equina diseased. Degenerated fibres in trunk of vagus nerve.

Diagnosis.—Clinically and anatomically typical tabes, together with syphilitic cerebro-spinal meningitis.

He alleges that this is the first case precisely of the kind, and that whilst Oppenheim's and Eisenlohr's cases simulated the image of tabes to a certain degree—or, as we have already seen for the cases of the latter, very closely—there was, in them, gummy meningo-myelitis, and a projection of gummy substance into the posterior horns (not a systematic disease).

He makes out his case to be one of such as bone-gummata *plus* tabes, or as syphilitic meningitis *plus* bulbar nuclear disease. In the former association the tabes; in the latter, the bulbar nuclear disease; being *not at all specific* in themselves.

As for ætiology, just as many agents:—alcohol, lead, gout, malaria, or syphilis, may have ætiological relations to atrophic granular kidney; so, also, for the ætiological relations of tabes.

In a complex case,[1] presenting various syphilitic cerebral and spinal lesions, the latter consisted of circumscribed syphilitic spinal pachy-meningitis; spinal lepto-meningitis; gumma of cord; lesion of all systems of nerve bundles of cord, more systemised in the lower dorsal and lumbar regions, and here recalling to mind the lesions of tabes. The spinal blood vessels were diseased.

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[1] Dr. Martin Brasch, *Neurologisches Centralblatt*, 1891.

In another syphilitic case,[1] among other lesions, was degeneration of Goll's columns and posterior dorsal nerve roots, and probably of an oculo-motor nerve.

Here it is well to remind the reader of the second case of F. Raymond, summarised in Part I. of this communication, in which there were, simultaneously, the lesions of general paralysis, of "legitimate" tabes, and of a superadded diffuse embryonic meningo-myelitis, chiefly, but not exclusively, affecting the posterior columns. This last-named lesion as well as that (cerebral) of the general paralysis are taken by their reporter to be syphilitic.

Here, also, must be mentioned E. Siemerling's case of congenital cerebro-spinal syphilis quoted, in abstract, in Part II. of this communication. The gait was ataxic.

There were severe intracranial syphilitic lesions, including those which had damaged, or led to degeneration of, a number of cranial nerves.

In the cord, throughout the spinal pia, especially at its middle and lower part, was marked gummatous growth, invading the substance of the cord itself irregularly with projecting outgrowths here and there. The posterior columns were lesed almost throughout the length of the cord, and were so, partly by direct gummy infiltration, partly and secondarily by compression. They were severely and very extensively affected in the dorsal region, in which, also, the posterior portions of the lateral columns suffered more or less severely. The spinal veins and arteries also were diseased. The nerve roots were but little affected. To sum up, there were gummy inflammation and neoplastic formations in the spinal soft meninges, invading the cord extensively, especially the posterior columns, which were much degenerated and altered. And during life, ataxic gait, and damage to, and degeneration of, many cranial nerves.

Some cases by Dr. McCall Anderson[2] illustrate tabetiform symptoms produced by spinal syphilis. In one case was paraplegia; and tabetiform symptoms in the early stages developed somewhat rapidly. Soon those were, to a large extent, lost in the signs of very diffuse spinal bulbar (and cerebral?) lesions (absolute paraplegia, universal and complete anæsthesia, lost reflexes, very marked dysphagia, &c.). In the other two cases, the taboid symptoms formed very rapidly, namely, in two weeks in the one, in four weeks in the other.

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[1] Dr. Richard Schulz, *Neurologisches Centralblatt*, 1891.

[2] *Op. cit.*, p. 38-46.

Cases like the above have been termed by some "syphilitic pseudo-tabes;" the clinical aspect in them is produced by specific luetic processes in the posterior spinal columns and posterior parts of the spinal cavity.

It is a striking fact that before any close relationship between syphilis and tabes had been generally admitted, the description of certain ocular symptoms in each presented close similarity or identity. For not only general internal ophthalmoplegia, but also loss of light-reflex only, or of accommodation only, has long ago been found dependent upon syphilis; while in tabes the reflex iridoplegia to light only, was accepted as being commonplace.

Dr. L. Chabbert[1] reports "a case of general paralysis, of the form of tabes at the onset, in a syphilitic person." A married woman, aged 43, had incurred a sore at the age of 24, when her husband had a syphilitic rash. But she had no further manifestations whatever of syphilis. Her father and his brother and sister were more or less insane; her mother drunken; she herself, in childhood, timid, fearful; in youth, hysterical and mystical; and throughout life refractory to the simplest process of education, and of defective energy and will power. Heredity being the predominant factor, syphilis, if it really existed, may have acted on that basis. In the absence, as yet, of death and a necropsy, little can be gleaned from the case on this point, or on that as to the relations between tabes and general paralysis. And, indeed, the only signs raising a suspicion of tabes were some seizures, in the prodromal stage, somewhat like gastric, vesical and rectal crises. Such crises are not unknown in general paralysis. And not infrequently are there cases of general paralysis with some of the early symptoms far more like tabes than in the above case.

ADDENDUM.

Dr. A. Boettiger[2] describes, at length, a case of chronic gummy hereditary syphilitic meningo-myelitis affecting spinal cord, cerebellum, pons and bulb, &c.; the cerebellar symptoms and lesions (chiefly tumour) being very prominent.

A girl, aged 9 years, suffered eight months before admission, from sudden symptoms of brain pressure, which ceased in three weeks: from two to three months later came ocular paralyses, ceasing after one and a-half to two months; during the same time were visual and auditory disorders, and three attacks of loss of consciousness.

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[1] *Archives de Neurologie*, 1894, p. 401.

[2] *Archiv für Psychiatrie*, Bd. xxvi., Heft 3.

On admission, were choked disc with incipient atrophy; very sluggish reaction to light by the dilated pupils; complete deafness on right side, and on the left for tones; gustatory disorder; irregular pulse. Later on, besides symptoms of brain pressure, were weakness of lower limbs, enuresis, painful stiffness of nape, monospasms, hemispasms, and general convulsions of cortical type, extreme constipation; right knee-jerk absent, left lessened; severe attacks of pain in legs, more seldom in arms; spastic phenomena of the same distribution, at first as paraspasm, later as persistent contracture. Finally, rapid pulse, vagus paralysis, increasing marasmus, broncho-pneumonia, death five and a-half months after admission. Intelligence and sensibility apparently had not been much affected.

Besides some cranial changes, were cohesion of pia and brain cortex, internal hydrocephalus, ependymitis, occlusion of fourth ventricle by a tumour of the inferior vermis, of meningitic origin, coherent with and injuring the floor of the ventricle; chronic meningitic changes about the brain-base and nerve exits, diffuse nuclear infiltrations in pons and bulb, flattening of olfactory bulbs, altered optic nerves, degeneration slight in third nerves, extreme in cochlear nerves, partial in the vagi-glosso-pharyngei. Cerebellar meningitis and diffuse infiltration of gyri. Extreme chronic spinal, and especially lepto-, meningitis, more marked posteriorly. Growths consisting of small cell, or nuclear, accumulations jut from the meninges into the posterior and postero-lateral columns of cord—or lie free within—and destroy nervous elements; thickened glia fibre feltwork in same parts; diffuse small cell—or nuclear—infiltrations in grey and white. Nerve cells and fibres and roots degenerate. Heubner's change; and hyaline degeneration, of vessels.

The brain-pressure symptoms and optic atrophy can be assigned to the cerebellar tumour, the occlusion of fourth ventricle and secondary internal hydrocephalus: the transitory squint and third nerves' degeneration may be correlated with the morbid basal process; the disorders of smell and hearing are explained by the changes found in the olfactory bulbs and acoustic nerves; the pulse anomalies by those in the nuclei and roots of vagus: the spasmodic and convulsive symptoms may have been due to the increased cerebral compression, or to the infiltration of the cortex; the stiff nape is connected with the cervical meningitis, the limb pains with the infiltration of posterior spinal nerve roots and columns, the spastic phenomena with the growths (cell-heaps) and destructive changes in the posterior and lateral columns and infiltration in anterior horns.

The muscular laxity and defective tone are correlated with the neoplasm and other cerebellar lesions; so also is the failure and loss of knee-jerk: for in explanation of this pseudo-paralysis the views of Luciani on the cerebellum are adopted. As to the failure of knee-jerk, the cases and doctrine of Bastian as to total transverse spinal lesion; the doctrine, as to withdrawal from cerebellar influence, of Jackson; and other like cases on record, including that of Bruns; as well as cases of cerebellar lesions, especially tumours, as in an example by Handford;[1] all go to show that the knee-jerks may be abolished by conditions concerning the cerebellum; and that the tumour cases in which this occurs are (or are chiefly) those of the inferior vermis, or thereabouts.

(To be concluded in the next number.)

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[1] BRAIN, 1892, p 458.

ON THE CONDITION OF THE REFLEXES IN TOTAL TRANSVERSE DIVISION OF THE SPINAL CORD.

[*With a Critical Digest.*]

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I.—Introduction. The Reflexes in Cerebral Disease.

The study of the deep[1] and superficial reflexes is one of the greatest importance in the physiology and pathology of the nervous system. For many years we have been taught that the reflexes whose arcs existed in the spinal cord, not only of animals but also of man, were controlled or inhibited by centres in the brain, and we have used this knowledge to a large extent in the elucidation of many nervous disorders. It has generally been laid down as an almost absolute rule that when these controlling brain centres were cut off, either by a brain or a cord lesion, from the reflex centres in the cord, an increase of the reflexes, together with a spastic condition of the legs, resulted. Thus, whether we had to deal with a brain tumour or hæmorrhage, or with any disease or injury of the spinal cord, which interfered with certain efferent fibres (the pyramidal tract in the case of deep reflexes), we had increased reflexes in those parts of the body supplied by the cord at a lower level than the lesion, provided always that the spinal reflex centre itself was intact.

Several peculiarities were, however, noted in connection with this. As regards brain lesions, whether in animals or in man, it was found that immediately after a sudden injury of the cortex or pyramidal tracts the muscles supplied by the parts affected became flaccid, and all reflexes were absent; subsequently, however, the deep reflexes gradually returned in the course of a few hours or days, and finally became excessive, with marked rigidity of the muscles. The superficial reflexes, however, often did not return, but remained permanently absent on the paralysed side.

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[1] More accurately known as myotatic contractions. The term "deep reflex" will be used, however, throughout this article for the sake of simplicity.

The theories generally put forward to account for these phenomena are as follows:—The initial loss of the reflexes is usually described as due to "shock," just as may occur in shock from injuries in other parts of the body. The subsequent increase of the deep reflex is said by some to be due simply to the cutting off of the restraining influence of the cortex; but this explanation is not sufficient, as it does not account for the gradual onset of the increase. The explanation of the French school, as put forward by Bouchard, Charcot and Brissaud, is that the rigidity and increased reflexes are only an effect of the sclerotic myelitis of the pyramidal tracts which occurs in the course of their secondary degeneration, and Brissaud assumes that the irritation of their fibres by the overgrowing neuroglia would keep up a continuous condition of irritation in the ganglion cells. This is not satisfactory, however, as increased reflexes and even ankle clonus may occur in hysteria or after attacks of Jacksonian epilepsy, and Bastian (1) mentions the case of a girl with marked increased reflexes from Pott's disease, in whom, after death, no descending degeneration of the pyramidal tracts was to be found.

Gowers (2) says the gradual increase of the deep reflex may be due to the lesion of the pyramidal tracts, causing a degeneration below at the termination of the pyramidal tracts in the grey matter. The excess of action of the spinal centres may be the result of a degeneration of the terminal structures which may form a controlling mechanism which normally restrains the activity of the muscle reflex centres. The interval which elapses before the excess is manifested may be due to the fact that there is little tendency to increased activity; that but slight control is habitually exercised or needed; and that the capacity for over action is gradually developed and gradually increased.

Hughlings Jackson (3) says the increased reflexes are due to the cutting off of the cerebral influence, thus allowing the unantagonised cerebellar activity to manifest itself.

Bastian (1) elaborates this theory, and while admitting that the increased reflexes are due to an increased muscular tone, supposes that muscular tonus is due mainly to cerebellar influence acting upon and through the spinal centres, and the removal of cerebral influence may allow cerebellar influence to reach the cord more freely; that is, there is an unrestrained (not unantagonised, as Hughlings Jackson puts it) influx of cerebellar energy. This view will be again alluded to later. Bastian says, however, that it is not easy to explain on this theory why the increase in the reflexes manifests itself slowly and does not immediately follow the cerebral lesion. He says it may be that in such cases the extra leakage of cerebellar energy has a tendency to go on increasing up to a certain point because of the gradually lessening resistance (probably in the pons) opposed to any such overflows of cerebellar molecular energy. All nerve actions, whether normal or abnormal, become easier and recur all the more readily, the more frequently they are repeated.

As regards the superficial reflexes in hemiplegia, Gowers (2) suggests that in man the higher controlling centre is the optic thalamus, and that this is itself under the habitual control of a higher centre, perhaps in the cortex. When this last is destroyed or the path from it is interrupted, the controlling centre in the optic thalamus passes into a condition of increased activity, and the reflex action is permanently lessened, thus accounting for the frequent permanent lessening of the superficial reflexes in hemiplegia. An objection to this is that the deep reflex is said to depend on the muscle tone, which, according to Mommsen (4) is in its turn dependent on a muscle reflex action (true reflex) excited by tension acting on the sensory muscle nerves and Heidenhein's experiments show that tone depends on tension and is a reflex process. If this is so then, presumably, though not of course certainly, this reflex would also be controlled by the optic thalamus and the tone, and (consequently the deep reflex) would be absent in hemiplegia, which is not the case.

II.—The Reflexes in Disease and Injury of the Spinal Cord.

Next we must examine the views held as to the condition of the reflexes in spinal cord injury or disease.

It is admitted by all that if disease of the spinal cord merely affects the pyramidal tracts the superficial and deep reflexes below the lesion are increased and the muscles are in a state of rigidity provided that the reflex arcs are themselves healthy. Also if the cord is injured (as by a crush) but is not completely divided, then a similar condition as regards reflexes and muscular rigidity is admitted.

As the result of an injury totally dividing the cord, either in animals or man, it has always been taught and is still held to be true by many of the best observers, that after a period of shock during which the reflexes are absent, the reflexes supplied by the parts below the injury gradually increase until they become excessive and great muscular rigidity sets in, a condition similar in fact to that which occurs in brain lesion.

Experiments on Animals.

In the lower animals, if the spinal cord is cut off from the brain, we have undoubtedly first a period of shock, during which the reflexes are absent and the muscles are flaccid. But examining such animals as the frog, dog and rabbit, we find that in the frog the period of shock is a very short one and that the subsequent reflexes of the isolated cord are very complex and quite purposeful in character; in the dog the period of shock is much longer and the subsequent reflexes not quite so purposeful, while in the rabbit the reflexes are still less complex.—Foster (5).

Reichert (6) made some experiments to ascertain the effect of cerebral disturbances on the knee-jerk. He cut completely across (as proved by *post-mortem* examination) the spinal cord of a dog in the lower cervical region. Immediately after the section the reflexes were diminished, but in the course of two hours or even less they were normal.

Ferrier (7) completely divided the spinal cord of a monkey at the level of the eighth dorsal root. "Immediately after the operation the knee-jerks were as capable of being elicited as before the operation. The animal exhibited the usual paraplegic symptoms, both as regards total paralysis of motion and sensation, and continued so until its death, four months later. During this period there gradually developed well-marked increase of the knee-jerks, as well as of the superficial reflexes, together with considerable rigidity of the limbs." In the second case the cord of a monkey was divided at the level of the sixth dorsal root, and both knee-jerks were present immediately after the operation. The animal lived for two months. Twelve days after the operation the knee-jerks could not be obtained and remained absent for sixteen days, during which the muscles wasted, especially on the left side; then the right knee-jerk re-appeared and increased; the left was not further examined before death.

T. S. Risien Russell's experiments (8) show that the immediate effect of complete transverse section of the cord above the level of the lumbar enlargement in the dog depends on the depth of narcosis. "The cord was always divided at or about the mid-dorsal region, and in no instance did the knee-jerk disappear after such section of the cord when there had been no doubt as to its presence before the lesion was inflicted, while usually there was no difficulty in being certain of its increased activity under such circumstances. The depth of the narcosis, however, plays a most important part in this connection, for if the cord were divided when narcosis was so profound that the knee-jerk was almost abolished, the section of the cord hastened the result; and when the cord was divided, when the depth of narcosis was such that the knee-jerk was abolished, the latter remained absent for a much longer time than is usual after suspension of the administration of the anæsthetic when the spinal cord has not been divided."

Observations in Man.

When we come to man, however, there is a considerable diversity of opinion concerning the reflexes in total transverse division of the cord. As I have said, many insist on the existence in these cases of the condition mentioned above, provided that the reflex arcs themselves are normal.

Dr. Bastian (1), however, in "Quain's Dictionary of Medicine" in 1882, in his work, "Paralyses, Cerebral, Bulbar and Spinal," published in 1886, and later in an article in the *Medico-Chirurgical Transactions* for 1890, brought forward several cases of total transverse softening of the spinal cord in the cervical or upper dorsal regions in which he states there was total absence of both superficial and deep reflexes in the legs with a flaccid paralysis of the muscles; there was but a slight muscular wasting and very little, if any, diminution in the degree of muscular contraction to the faradic and galvanic currents. "For the first ten days or fortnight there is often complete retention of urine, but after some time when the lumbar region of the cord again becomes capable of manifesting to some extent its centric functions, the initial retention gives place to incontinence, the urine may now be discharged at intervals of two or three hours in small quantities owing to the occurrence of reflex contractions of the bladder when it attains a certain degree of fulness. The passage of a catheter, however, will often show that the bladder is never completely empty, two to four ounces of urine remaining after the reflex contractions. The bowels are usually constipated and relieved only after the administration of aperients or by enemata. At these times there is generally incontinence of fæces, the patients having no power of controlling the reflex actions concerned in defæcation, when they have once been strongly excited." In Bastian's earlier work he says that in these cases "nearly all the reflexes dependent upon the lower portions of the cord are abolished; the muscles of the lower extremities may show some slight irritability when they are forcibly tapped, and when the soles of the feet are strongly tickled there may be very slight movements of the toes; but beyond this there is often an entire absence of all reflex movements." In his last paper however, Bastian states that all deep and superficial reflexes are lost, but that the organic reflexes (those of the bladder and rectum) may be retained.

In the *Medico-Chirurgical Transactions* he mentions four cases. In the first, a woman who had previously suffered from scirrhus of the mamma which had been removed, gradually developed total anæsthesia and paralysis below the ensiform cartilage with flaccid muscles and absence of all superficial and deep reflexes, retention of urine, and fæces passed unconsciously. Later the muscles of the legs contracted when tapped with a stethescope, and there was a slight plantar reflex when the soles of the feet were sharply tapped. At the *post-mortem* examination there was found an angular curvature of the fourth and fifth cervical vertebræ from softening of the bone, together with a total transverse softening of the cord for three-quarters of an inch, near the middle of the cervical swelling; there was ascending secondary degeneration in Goll's columns and the superficial lateral tracts, descending degeneration in both crossed pyramidal tracts and anterior columns and in the comma-shaped tracts; but all through the lumbar swelling and in the lower half of the dorsal region the grey matter presented a normal appearance.

In his second case, there was cancer of the vertebræ in the upper dorsal region secondary to cancer of the breast, with pressure and softening of the cord in the same region. When the paralysis of sensation and motion were thoroughly established in the legs, there was an absence of all reflexes except that forcible tapping of the soles caused slight movement of the toes; there was unconscious action of the bowels and bladder, but the urine was passed in gushes at frequent intervals.

The third case was a man with total transverse softening in the mid-dorsal region; before the loss of sensibility and motion was complete his reflexes were increased, but as soon as complete the knee-jerk, ankle clonus, cremasteric, abdominal and epigastric reflexes disappeared, but the plantar reflexes were present on both sides, and later became exaggerated, and occasional twitching of the muscles of the legs occurred; the urine dribbled away at short intervals. The plantar reflex was almost abolished on the day of death. Bastian thinks that sensibility was not quite lost in this case because of the twitching which occurred (but this is rather begging the question). The *post-mortem* appearances were softening at the point mentioned with the usual ascending and descending degeneration. The lumbar grey matter looked normal but pale.

In the fourth case, that of a man with tubercular caries of spine with softening of the cord, when the loss of motion and sensation was complete there was an absence of all reflexes, but tickling the soles of the feet and pricking of the eyes, although not felt by the patient, caused starting movements; the urine was voided in small quantities at frequent intervals; the electrical reactions were normal. After a time the sensation and motion slightly improved, and with this there was a slight knee-jerk on the left side with very slight ankle clonus, and then these were found on the right side also. The patient became worse with total loss of sensation and motion in both legs, together with loss of knee-jerk and ankle clonus on the right side, and very slight on the left, but plantar reflex was present. Before death all reflexes disappeared. *Post-mortem*, the cord was found quite softened in the upper dorsal region with the usual secondary degenerations. but the lumbar grey matter was normal. The length of life of these four cases was eight weeks, seventeen weeks, nineteen days and seventeen weeks respectively.

Dr. Bastian then refers to several other cases in which either from disease or injury the cord was totally divided, and these I will allude to briefly.

The first is a case of Dr. Gee's, in which a man was attacked with paraplegia coming on in two days, and causing paralysis of motion and sensation up to the level of the third dorsal nerve. There was total loss of the superficial and deep reflexes, but on one occasion the knee-jerk returned very slightly; this again disappeared but returned again, and the plantar reflex became well marked, but at the same time there was a slight return of sensation in the legs. Later, however, when the anæsthesia was complete, there were involuntary movements of the legs, causing them to be completely flexed, and they moved with the least irritation of the skin. The electrical reactions were normal. At the *post-mortem* examination there was complete softening of the cord at the third dorsal level, but the grey matter of the lumbar region was normal.

Tooth (9) mentions a case of fracture dislocation of the fifth and sixth dorsal vertebræ, with complete transverse crush of the cord, the man living for twenty weeks and four days. There was total paralysis of motion and sensation up to the sixth rib. No reflexes could be obtained except the epigastric on the right side. Two months after admission it was noted that on pinching the lower part of the thigh the ham-string muscles contracted, but there was no sole reflex.

Kahler and Pick (10) report the case of a man with fracture dislocation of the sixth cervical vertebra. The leg muscles were quite flaccid but not wasted, and there was total paralysis and anæsthesia; all reflexes below the lesion were abolished, excepting the plantar, which was very weak. Death occurred on the seventeenth day. The lower parts of the cord were healthy.

Willett (11) mentions a fracture dislocation of the seventh cervical vertebra with absolute paralysis of motion and sensation, with flaccid muscles and complete loss of all reflexes until death, six months after. The cord was examined by Tooth, and the lumbar region was normal, except for the usual descending secondary degenerations.

Willett reports another case where there was a fracture dislocation of the fourth cervical with complete paralysis of the lower limbs and complete anæsthesia below the fifth rib. There was a total absence of tendon reactions in the arms and legs; death occurred six weeks afterwards, and neither sensation, motion, nor reflexes returned. The lumbar region of the cord was healthy except for the usual secondary degeneration.

Langton's case was a fracture dislocation of the sixth cervical vertebra with absence of the reflexes. Three weeks after the injury the sole reflexes were well marked, and the right knee-jerk had returned, but was feeble; the left was not tried. Tooth also examined this cord and found it completely crushed, but Weigert's method was not used. Bastian says this is not a very conclusive case, but that, at any rate, the reflexes were not exaggerated.

Bowlby (12) examined the condition of the reflexes in twenty-one cases of complete crush of the cord, as verified by *post-mortem* examination, and in three cases of partial injury to the cord. In ten cases of complete crush the patients only lived a few hours, during which there was no rigidity of the muscles and the reflexes were not present. These cases he does not further consider, as they only lived such a short time and shock could not be eliminated.

In the remaining eleven cases of complete crush, shock could be eliminated almost entirely, as they lived for periods varying from three days to ten months. In all there was total paralysis of sensation and motion below the lesion, the urine was retained and the fæces passed unconsciously. The deep reflexes were permanently lost in all the cases. The superficial reflexes were also generally lost immediately after the accident, although this was not invariably the case, and they, unlike the deep reflexes often returned in the course of time. In Case 2, the reflexes were never lost; in Case 3, they returned on the eighteenth day; in Case 4, not until the twenty-second week; in Case 5 (when the patient survived five months), they were not noticed to return, and this also happened in all the other patients who survived for periods varying from one day to six weeks, with the exception of Case 11, in which the sole reflexes returned on the second and third days following the injury.

In the three cases of partial paralysis of motion and sensation, the muscles were in a state of rigidity and the reflexes were all increased.

Bowlby concludes by agreeing with Bastian as to the loss of the deep reflexes, but says that the condition of the superficial reflexes requires further investigation. As can be seen from his cases they are certainly not always lost.

Thorburn (13) has re-examined his cases since Bastian published his paper in 1890, and concludes that in total transverse lesions of the cord from injury, both the superficial and deep reflexes are permanently and entirely abolished, that it is only in partial lesions that there is retention or exaggeration of these reflexes, and that the abolition of the reflexes is a result of isolation of the spinal centres from their cerebellar connections. His observations were limited to testing the tactile sensations, the other sensations not having been tested. All the cases were due to injury, in all except two the injuries were above the mid-dorsal region and in all (although no microscopic examination was made) there was no evidence that the lumbar region was affected. He reports the condition of nineteen cases with complete division of the cord, and ten with incomplete division. The lesion was supposed to have been complete when paralysis and anæsthesia were totally below its level and incomplete when there was at any period some sensation or some motor power below.

Of the complete lesions he records the condition of the superficial and deep reflexes in nine, of which eight were observed from the commencement. In all the deep reflexes were absent the whole time and the superficial also, except in two cases, in one of which the plantar was present and exaggerated (but the patient was suffering from the effects of drink and only lived forty hours, so that anæsthesia may not have been complete); the other was observed for some months, and after a few days the plantar and cremasteric reflexes were present but the epigastric was lost (the man, however, was partially demented). The effects in these cases could not have been due to shock, as the last observations were made after forty hours (two cases), two days, four days, ten days, twenty-five days, three and a half months and five months, after injury, in the respective cases.

In four cases of incomplete lesion the injury was slight, sensation was not affected and the reflexes were exaggerated. In another case there was slight power of moving the limbs and anæsthesia up to the sixth rib, the knee-jerks and plantar reflexes were exaggerated, the cremasteric and abdominal were absent; the patient gradually got worse and before he died in five weeks all reflexes disappeared. In another case paralysis came on about ten minutes after the accident; paralysis was at first complete and anæsthesia nearly so, and all the superficial and deep reflexes were absent. On the fifteenth day, there was a distinct knee-jerk on the right side and a slight one on the left, and the passage of a catheter, though not felt, caused reflex contractions of the sartorius.

In another case, one of fracture of the third dorsal vertebra, there was complete paralysis and anæsthesia with entire loss of reflexes which remained for about ten days; then subjective sensations appeared in the feet with ability to tell when the bladder was distended, and at the same time a feeble knee-jerk appeared on the right side only; a year after there was total paralysis and anæsthesia, disappearance of the knee-jerks, but slight plantar reflex on the right side.

Thorburn summarises as follows :—" (1) Shock is not the cause of early loss of reflexes in spinal injuries, as such shock is just as great in cases in which these are retained as in those in which they are lost. (2) When the lesion causes complete paralysis and anæthesia the deep reflexes are always lost.[1] (3) When motor or sensory power (or both) return, in even a slight degree, these reflexes also return. (4) When no such recovery occurs, the deep reflexes remain absent for periods of practically indefinite duration. (5) When the lesion does not cause complete paralysis and anæsthesia the deep reflexes remain in either a normal or an exaggerated form. (6) But if the motor and sensory symptoms afterwards become complete, such reflexes disappear. (7) The superficial reflexes, however, do not conform absolutely to these rules. In nearly all cases, both of complete and partial lesions, these disappear and remain absent, the 'plantar reflex' only existing. The latter is obtained in many cases of complete and partial lesions."

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[1] This statement I shall refer to later.

Thorburn thinks also that the visceral reflexes conform to the same rules, and points out that Bastian's statements in regard to them would certainly support the "shock theory." He says the subject is a very difficult one, as "incontinence of urine" may mean mere overflow or active but involuntary discharge. The escape of the urine "in gushes" at distinct intervals (which most observers seem to think indicates a reflex emptying of the bladder) may be due to contraction of the abdomen and diaphragm, to external mechanical pressure on the abdomen, to changes in the position of the patient, to gaseous distension of the intestines, or to contraction of the bladder walls set up by external pressure. As a rule in all cases there was permanent retention unaccompanied by any evidence of reflex activity; in cases with retention of the body reflexes there was no retention of urine.

The condition of the rectal reflex, as judged by the fæcal discharge, is even more difficult to ascertain, as the fæces are passed less regularly and their discharge is governed, not only by the condition of the rectal reflex centres, but also by the consistency of the intestinal contents, the activity of peristalsis, and the contractile power of the voluntary muscles of the abdomen. In complete lesion there may be either retention or discharge of fæces, whereas in partial lesions the action was more normal. [No observer seems to have used Gowers' simple method of testing the reflex of the sphincter ani, namely, that of inserting the finger and feeling if contraction ensued.]

Ferguson (14) records the three following cases: (1) Angular curvature at the fourth dorsal vertebra. At first the knee-jerk was exaggerated, but gradually diminished and disappeared; there was no atrophy beyond that of disuse, nor was there any reaction of degeneration; there was complete paralysis and anæsthesia *Post-mortem*, there was found complete softening of the cord at the level mentioned, but the lumbar grey matter was normal. (2) Fracture dislocation between second and third dorsal vertebræ. The knee-jerk at once disappeared and remained absent for eighteen months. The bladder reflex was fairly good; there was no muscular atrophy or reaction of degeneration; priapism was present but the legs were not rigid.

Post-mortem, the lumbar enlargement was normal, except for the usual secondary degenerations. (3) Dislocation of the sixth and seventh cervical vertebræ. The knee-jerk was permanently absent, though the patient lived for five weeks; the bladder acted fairly and priapism was present; no reaction of degeneration or atrophy. *Post-mortem*, the cord was thoroughly destroyed at the point mentioned.

Livingston Davies (15) mentions a case of fracture of the fifth cervical vertebra with immediate paralysis below the lesion, and absence of knee-jerks, but the case only lived six hours.

Westphal (16), as early as 1870, in a case of sarcoma of the dorsal vertebra, mentions that the tendon reflexes were not increased, and Leyden (17), in 1878, relates a case of tumour of the cervical cord in which the reflexes were absent; and in 1892 (17), a case of total transverse gonorrhœal myelitis in the dorsal region, with absence of the deep reflexes, but the cremasteric was occasionally present.

Herter (18) publishes five cases bearing on this subject.

Case 1.—Dislocation of sixth cervical vertebra. Total loss of motion and sensation, with absence of knee-jerks and all superficial reflexes; the urine and fæces passed unconsciously. In three days the cremasteric reflexes reappeared. Death occurred on the sixth day, and, *post-mortem*, total destruction of the cord with hæmorrhage in the grey matter, principally at the eighth cervical root, was found.

Case 2.—Dislocation of sixth and seventh cervical vertebræ with paralysis of motion and sensation below the lesion, loss of the knee-jerks, cremasteric, abdominal and plantar reflexes, and the urine and fæces passed unconsciously. Death in forty hours. *Post-mortem*, there was found destruction of the cord at the eighth cervical segment.

Case 3.—Fracture of the third cervical vertebra with complete paralysis and anæsthesia, loss of the knee-jerks and of the cremasteric, plantar and abdominal reflexes. Death on the same day.

Case 4.—Fracture, dislocation of the sixth cervical with paralysis and anæsthesia below the lesion, total abolition of the reflexes, superficial and deep, retention of urine and incontinence of fæces; death in two days.

Case 5.—Partial injury to the cord with loss of power in the arms, especially the left, and partial anæsthesia of the inner side of the left arm and the left side of the trunk. The legs were normal in all respects; the knee-jerk was increased on the left side, and ankle clonus was present. On the right side the reflexes were normal.

The reviewer (19) reported a case of injury to the spine in which, ten months after the accident, there was total paralysis and anæsthesia below the seventh dorsal nerve, with absence of knee-jerk and ankle clonus, but marked exaggeration of all the superficial reflexes, except the epigastric, which was absent. There was some wasting of the muscles of the legs, but the electrical reactions were normal. The urine was voided in gushes at distinct intervals. *Post-mortem* (patient died in June, 1894), there was found a complete crush of the cord from fracture dislocation of the sixth dorsal vertebra.

Hughlings Jackson (20), in speaking of Bastian's theory, says: "So far as I have observed in cases of fracture dislocation, crushing the cervical cord completely across, the knee-jerks are lost. . . . It may be said that, did the patients referred to live long enough, their knee-jerks would return. In some dorsal cases under my care the knee-jerks have returned, in one case after being absent for thirty-eight days, and in another for two years; there is in neither case any return of sensation or motion in the legs. . . . . It has been said that when in chronic cases the knee-jerks are absent, there are set up secondary morbid changes in centres below the lesion, and perhaps in peripheral nerves; we have, however, to explain not only the loss of the tendon reaction, but also the presence of the superficial reflexes. . . . . Moreover, those who suppose that secondary morbid changes set up in centres below the lesion cause loss of the knee-jerks for months, have to account for the return of these tendon reactions in some cases."

The following cases of the condition of the reflexes in tumour affecting the spinal cord, are taken from cases given in Gowers' and Horsley's lists:—

Vincent Harris (21). Sarcoma of the periosteum of the laminæ of the sixth, seventh and eighth dorsal vertebræ with complete softening of the cord; complete paralysis and anæsthesia. Reflexes all exaggerated at first, but completely lost later.

Bruce and Mott (22). Myxoma near fifth dorsal segment, cord completely disorganised at the point, complete paralysis and anæsthesia; knee-jerk increased at first, especially the left; later lost, and then reappeared in both feet, returning in left first. Later, frequent spasmodic twitchings in both legs. Plantar reflexes present and exaggerated throughout. Cremasteric and abdominal absent. Epigastric present, later absent; general emaciation, but no special wasting in the legs; some diminution of response to both currents.

Gowers and Horsley (23). Myxoma of dura mater between third and fourth dorsal nerves on the left side; cord severely indented, probably with ascending and descending degenerations; complete paralysis and anæsthesia, paralysis of bladder and rectum; all reflexes much exaggerated, very severe spasms, very marked rigidity usually in extension, general emaciation. Recovery by removal of growth. (See appendix.)

Traube (24). Sarcoma inside dura mater in cervical region, and cord softened from bulb to seventh cervical. Complete paralysis and anæsthesia. Plantar reflex weak, painful spasms, especially in lower limbs, and finally contracture.

Malmotte (25). Fibroma within dura mater growing from arachnoid between second and third dorsal vertebræ. Cord compressed but otherwise healthy. Complete paralysis and anæsthesia below lesion; paralysis of bladder and rectum. Great excess of all reflexes. (See appendix.)

Babinski (26) relates four remarkable cases, two observed by himself, one quoted from Kadner and another from Druschky. In the first there was a tubercular pachy-meningitis compressing the cord at the fourth and fifth dorsal vertebræ; there were found microscopically, only slight lesions at the point of compression, and the consecutive sclerosis of the lateral tracts was quite rudimentary. There was, however, complete flaccid motor paraplegia without muscular atrophy or reaction of degeneration, no anæsthesia, but with vesico-rectal trouble and bed-sore, and complete abolition of the knee-jerks.

The second case had purulent pachy-meningitis pressing on the cord at the fourth to the seventh cervical vertebræ, microscopic examination, however, showing the cord to be practically normal in all parts. There was weakness and atrophy of both arms; complete motor paralysis of both legs, but with no wasting of muscle or reaction of degeneration, complete flaccidity of muscles, absence of tendon reflex, no loss of sensation, but vesico-rectal troubles and a bed-sore. Death occurred in five months.

Kadner's case was one of carcinoma of the upper four dorsal vertebræ, with flaccid paraplegia and disturbance of sensibility, retention of urine and bed-sore. The motor paraplegia was complete during seventeen days, and during that time the reflex of the Achilles tendon was weak, and the patella reflex absent. There was no secondary degeneration of the pyramidal tracts.

Druschky's case was a compression of the spinal cord following an external meningitis between the second and sixth dorsal vertebræ, the foot clonus was absent, and there was no secondary degeneration.

These partial cases of Babinski's are unsatisfactory, as it is probable from the disease of at least three of the cases, that the cachectic condition of the patients caused the loss of the reflexes (peripheral neuritis ?).

Grasset (27) mentions a case of Pott's disease, with compression of the spinal cord followed by a flaccid paraplegia with complete anæsthesia of the lower limbs, and whose condition was much relieved by treatment, showing that the cord had not been destroyed at the point of compression (an important fact in the prognosis of these cases).

Mitchell Clarke (28) reports fully a case of fracture dislocation of the third and fourth dorsal vertebræ which lived for ten months. There was absolute loss of motion and sensation below the lesion, the urine and fæces were retained, and the plantar, cremasteric and abdominal reflexes and the knee-jerks were absent; there was no rigidity of the muscles, but they were flabby and small from disuse. These conditions remained until death. At the *post-mortem* examination, the cord was completely destroyed at the point of injury, and, excepting for the usual ascending and descending changes, was quite healthy, the grey matter of the lower dorsal and lumbar regions being entirely normal.

I have reserved until the last a paper of Bruns (29) on this subject. It is undoubtedly the most important which has appeared, because of the very exhaustive and elaborate microscopic examination of the whole cord, of the peripheral nerves and the muscles. As it is abstracted in the present number of BRAIN, I need not further mention it, except to say that it establishes without doubt that a total transverse division of the cord, without any affection of the lumbar grey matter or of the peripheral nerves, may be accompanied by a paralysis of motion and sensation, a lasting (death in one hundred and six days) absence of all reflexes, and a flaccid condition of the muscles below the lesion.

In his paper Bruns gives the following references to cases more or less similar, but which I have not been able personally to refer to: Pfeiffer (30) and Krause (31) in cases of cord tumour, Howard van Rensellær (32) in Caisson disease; Bruns (33) in a case of traumatic hæmatomyelia in the cervical region with failure of reflexes for the eleven days during which the patient lived; and Walter Friedebergs (34) (nature of case not mentioned).

Foster, in the last edition (1890) of his "Physiology," makes the following wide generalisations: "When we turn to the teaching of disease (in man), however, we again find that reflex movements carried out by the cord or by parts of the cord are, on the whole, scanty and simple. . . . . Clinical experience shows that in man, when a portion of the cord is isolated, reflex actions carried out by means of that portion, so far from being exaggerated, are much more commonly exceedingly feeble or absent altogether. In the cases in which the physiological continuity of the lower with the upper part of the cord has been broken by disease, by some growth invading the nervous structures, or by some change of the nervous structures themselves, we may attempt to explain the absence from the lower part of co-ordinate reflex activity, such as is seen in the lower animals, as due to the disease, not only affecting the powers of the actually diseased part, but influencing the whole cord below, and either by inhibition . . . . . or in some other way depressing its functions. But the same absence of complex reflex movements is also often observed in cases in which the cord has been severed by accident, and indeed, though accidental injuries to the human cord generally produce more profound and extensive mischief than that which results in animals from skilful experimental interference, clinical experience tends, on the whole, to support the view that in man the more complex subordination of the spinal cord to the brain has led to the dying out of the complex reflex actions which are so conspicuous in the lower animals. This, however, cannot be regarded as distinctly proved."

Further, Foster states: "Though we know . . by an appeal to our consciousness, that an action of the brain, an effort of the will, may stop a particular reflex act, we have no evidence that in man separation of the cord from the brain leads, as in animals, to heightened reflex activity. In diseases, or injuries of the cord, reflex actions are, as we have said, sometimes exaggerated, but it is possible, and indeed probable, that the increase is due to the morbid processes producing a greater irritability of the cord itself, and not to the withdrawal of any inhibitory influences. In many cases, in perhaps the greater number, no exaggeration, but a diminution or even absence of reflex activity is observed; so much so that could we trust explicitly to clinical experience, we should be inclined to conclude that the scantiness of spinal reflex action in man was due, not to any pre-occupation of the cord by influences proceeding from a dominant brain, but to an inherent paucity of spinal reflex mechanisms."

Conclusions from Observations.

From the above observations we may at once conclude that the results of total division of the cord in animals are entirely different from the results seen in man when the cord has been divided by injury or disease. As Gowers says: "It is probable that in animals such spinal mechanisms (as co-ordination) are developed to a greater extent than in man, and care must be taken in drawing inferences from one to the other." And Foster remarks: "When we compare a rabbit, a dog, a monkey, and a man, the differences in the vascular, digestive and respiratory systems of the four, striking as they may appear, sink into insignificance compared with the differences exhibited by their respective central nervous systems. We need caution when from the results of experiments on dogs or rabbits, we draw conclusions as to the digestion or circulation of man, but we need far greater caution when from the behaviour of the isolated spinal cord of one of these animals we infer the behaviour of the intact spinal cord of man."

The cases that have been brought forward seem to show conclusively that if the cord is totally divided in man, the deep reflexes below the lesion are lost. This is certainly not what would have been expected, and it is undoubtedly a difficulty, but one which has to be faced and explained if possible.

Theories to explain Loss of Knee-jerk.

The "shock" theory may at once be set aside when we remember that these cases have continued to live for many months and yet the deep reflexes have never returned.

Gowers disposes of these cases by remarking that: "If primary myelitis or injury to the cord high up, is followed by abolition of the muscle reflex action in the parts below, it is the result of inflammatory damage to the lower part of the cord in which the centres are situated, generally by a descending myelitis. . . . Sometimes such descending inflammation is limited to the grey matter or to the posterior columns, . . . and is a very different thing from the descending degeneration of the pyramidal tracts." Further on, in criticising Bastian's cases, he says: "There was also loss of the skin reflexes and of faradic irritability with rapid muscular wasting—in short, all the indications of a lumbar myelitis. The most probable explanation of these cases is that the descending degeneration of the pyramidal tract has been more than usually irritative in nature, so as practically to amount to a parenchymatous inflammation, and that this has invaded the motor structures as a nutritional change, sufficient to abolish their function, without, however, destroying their form."

This explanation is not satisfactory in the majority of cases I have brought forward, because an examination of them will show that in most the superficial reflexes are not absent, but present and even increased (*vide infra*), that the electrical reactions when tested have been found normal, and that the muscles of the legs are but slightly wasted. Now if, as Gowers suggests, there was a nutritional change in the grey matter of the lower dorsal and lumbar regions, which was of such a nature as to abolish the deep reflexes, there ought also to have been absence of the superficial reflexes, wasting of muscles and alteration of electrical reactions.

Herter attempts to explain these cases by saying that there is certainly some inhibition of the deep reflex coming from above, and that it consists of irritative discharges from the cord itself immediately below the injury, and which are due to pathological processes going on in the nervous structures. The evidence in favour of this inflammatory irritative process going on, Herter says, is to be found in other symptoms produced by it, such as the pain and muscular contraction found in those parts supplied by the cord immediately above the lesion.

An objection to this theory is that (arguing from our knowledge of increased knee-jerks in brain lesions) it is the pyramidal tracts which take down inhibitory influences from the brain to the lumbar centres, and in partial injuries of the cord, or pressure on the cord from tumours in which there has been much irritation of the pyramidal tracts (with total loss of sensation and motion, but yet the cord not absolutely divided, as in Gowers' and Horsley's and Malmotte's cases), there has been an increase of the knee-jerks and not a diminution, which should have been the case if Herter's hypothesis had been correct.

Ferrier says: " The difference between the symptomatology of total transverse lesions of the cord in man and the lower animals is due to the greater independent vitality of the spinal centres in the latter as compared with the former. In these, total separation from the higher centres leads to such impaired vitality that they are no longer capable of manifesting that increased excitability which is the primary cause of the phenomena in question."

Bastian bases his explanation of these cases on the hypothesis of Hughlings Jackson, that when cerebral influence is cut off from the cord it allows cerebellar activity to manifest itself unantagonised. Bastian points out that if the cerebrum is alone cut off as by a cerebral hæmorrhage the knee-jerk is increased, but if both cerebrum and cerebellum are cut off, either by a large pontine or a cord lesion, then the knee-jerk is absent. He infers, therefore, that the presence of the knee-jerk is due directly to the action of the cerebellum in keeping up the tone of the muscles, the amount of which is increased because the cerebellum is unrestrained by the cerebrum. He mentions in support of this that in hemiplegia from cerebral disease those muscles which are bilaterally associated are not in a state of spasm, as they are still under the influence of the cerebral hemisphere of the opposite side, but that all other muscles are in a state of increased tone. Bastian believes "that the cerebellum is a supreme motor centre for reinforcing and for helping to regulate the qualitative and quantitative distribution of outward currents, in voluntary and automatic actions respectively." He also thinks that the cerebrum and cerebellum seem to be distinctly associated with one another during all attempts to perform new and unaccustomed movements; but in the production of automatic movement it seems probable that in proportion to their degree of automicity the cerebral share in their production becomes less and less, whilst that of the cerebellum becomes more and more preponderant.

Another fact which is in favour of Bastian's view is that in many cases of cerebellar tumour, particularly of the middle lobe, the knee-jerks are absent, but this is certainly not the rule; moreover, the knee-jerk may be absent at one time and at another may be present during the course of the same case; in some cases tumour of one cerebellar hemisphere has been associated with loss of the knee-jerk of the opposite side, and in other cases with loss on both sides.

Luciani (35), as a result of many experiments on animals, states that after extirpation of the cerebellum there is asthenia, atonia and astasia. If the lesion is unilateral, he says that the muscles on the same side of the lesion appear to him more flaccid, and that the leg occasionally gives way, as if the tone suddenly ceased. Ferrier (7) states that Luciani does not seem to have investigated the tendon reflexes in his animals, and further states that his own experiments made in conjunction with Dr. Turner, and also the experiments of Dr. Risien Russell, show that after cerebellar extirpation, complete or unilateral, or after section of its peduncles, the knee-jerks are rather increased than diminished. In Risien Russell's (8) experiments on dogs the knee-jerk on the same side was exaggerated when one lateral half of the cerebellum or when one half of the posterior part of the vermis was removed, and this increase persisted for weeks. Russell says this may be due to one half of the cerebellum exercising an energising influence on the opposite half of the cerebrum, in virtue of which it causes the opposite cerebral centres to hold in greater check the spinal centres of the side of the spinal cord corresponding to the side of the cerebellar lesion; or it may be that the one half of the cerebellum exercises a controlling influence on the spinal centres on the same side of the spinal cord. As the increase of the opposite knee-jerk, which is consequent on the removal of the cerebral hemisphere of one side, is not nearly so great as that brought about by the removal of the lateral lobe of the cerebellum on one side, and secondly, when one cerebral hemisphere has been removed, and the opposite lateral lobe of the cerebellum is removed, the knee-jerk on the opposite side from the cerebral, and on the same side as the cerebellar lesion, is further increased after the second operation. Russell thinks that the cerebellum exercises an independent action on the spinal centres from that exercised by the cerebrum, and that this action is of the nature of a control over the spinal centres chiefly of the same side.

As we have already seen that experiments on animals do not help us very much in studying the subject in hand, we need not dwell further on them.

Bastian considers that the impulses which he imagines maintain muscular tone, may pass from the cerebellum to the cord by the "comma" tracts, or else diffusely through the grey matter in the same way that impressions of pain are conveyed in the reverse direction. As regards the "comma" tracts, Bastian thinks it is unlikely that they carry the impulses, as they terminate principally in the upper half of the cord. He thinks the grey matter is a more likely channel, because so long as sensation is intact the reflexes are increased, and he considers that the slightest bridge of grey matter would be sufficient. Arguing from a case reported by Thorburn, he thinks the efferent fibres are very near those which conduct painful sensations, as compared with those conducting sensations of touch. Several cases are, however, recorded in which both analgesia and anæsthesia have been absent, and yet the knee-jerks have been markedly increased (*vide infra*).

Another objection to the view that impulses flow from the cerebellum to the cord to keep up muscular tone is that practically no efferent fibres are known to pass from the cerebellum to the cord. A few efferent fibres certainly pass to the opposite inferior olive by way of the external arcuate fibres, and we know that a lesion of one cerebellar hemisphere may lead to degeneration of the opposite olivary body; moreover, Marchi has described a tract which degenerates downwards in the antero-lateral region of the cord after extirpation of the cerebellum, which he derives specially from the middle lobe, but this is doubted by Ferrier. Too much stress must not of course be laid on the mere fact that we do not know any definite tract from the cerebellum.

The Condition of the Superficial Reflexes.

Bastian, in his original paper, states that the superficial reflexes are lost in total transverse division of the cord, equally with the deep reflexes, and attempts to explain the various contractions of the legs which occurred in his cases when the muscles were tapped, as due to idio-muscular contraction, and not due to reflex action at all.

An examination of other cases mentioned will show, however, that in the great majority many of the superficial reflexes were, after a time, not only present but increased, that of these the plantar was most commonly present, often the cremasteric, and more rarely the abdominal and epigastric. That such reflexes are not idio-muscular in nature is shown by the fact that in these cases they may often be elicited quite easily by a very slight touch with a pin point, and that it is not at all necessary to strike the muscular fibres to obtain them. In a small minority of the cases the superficial reflexes are stated to have been absent throughout, and these are very difficult to explain away satisfactorily. It must, however, be remembered that it is not always easy, even in a normal individual, to obtain all the superficial reflexes at any one time. There are, in fact, many at present unknown influences affecting their absence and presence, quite apart from total transverse division of the cord.

Most observers now freely admit that in these cases the superficial reflexes are commonly present a short time after the injury has been received; it is, indeed, only another fact which shows the essential difference between the deep and superficial reflexes.

The Visceral Reflexes.

Most of the observers state that the urine is retained for long intervals but is passed "in gushes" at times. This at first seems to indicate that the spinal reflex for the bladder is fairly intact; that the accumulation of urine goes on for a longer time than usual, until, at length, impulses pass to the cord, which set in action the detrusor fibres and inhibit those of the sphincter. Thorburn has shown, however, that the voidance of urine "in gushes" may be due to many other causes principally mechanical in nature. If, however, there was no bladder reflex at work, and if the bladder muscles were all in that flaccid condition which is attributed to the leg muscles, then no great accumulation could occur in the bladder, which would be in a more or less "cadaveric" condition, and the urine would *constantly* dribble away. This does not seem to have been the case in the observation reported, so that it seems probable that the voidance "in gushes" is more reflex than mechanical in nature.

The bowels are generally stated to act unconsciously, but nothing more definite is known. As I have stated, the condition of the sphincter ani, as ascertained by inserting the finger, should be sought in all future cases.

The Condition of the Paralysed Muscles.

In all cases in which electrical examinations have been reported there has been no reaction of degeneration; in some the faradic and galvanic reactions are stated to be feeble, but in none are the faradic contractions absent, as one would have expected if the lumbar grey matter had been affected.

In a great number of the cases there is reported to be no wasting of the muscles, but in other cases wasting is noted. The term "wasting" has to be accepted with caution, as it may be a general wasting from the cachexia so frequently seen in these cases, and may not be that marked and rapid wasting seen in disease of the lumbar grey matter.

Again, in many cases the muscles are stated to be flaccid, but in others in a state of flexion. The latter condition is probably due in many cases to the irritation of bed clothes, &c., setting up reflex action, and may, therefore, be quite apart from the rigidity seen in cerebral or partial spinal lesions due to increased muscular tonus.

CONCLUSION.

From the above observations we must see that after total transverse division of the cervical or upper dorsal region of the cord there almost always results a lasting loss of the deep reflexes of the legs with flaccid paralysis of the muscles, and at the same time there may be no marked atrophy of muscles, no great alteration of the electrical reactions, and no affection of the grey matter in the lower dorsal and lumbar regions. No satisfactory explanation of the phenomena observed is as yet forthcoming, the cerebellar theory put forward by Bastian being perhaps the most feasible. More observations are yet necessary before this subject, which affects one of the most fundamental principles in neurology, can be satisfactorily settled.

APPENDIX A.

It has been stated by Thorburn, as a corollary to Bastian's theory, that when a spinal lesion causes complete paralysis and loss of sensation of the legs, the deep reflexes are entirely lost. Bastian, in speaking of the case of a young man, with an almost complete transverse softening in the dorsal region of the cord, associated with spinal caries and angular curvature with complete motor and sensory paralysis of the legs, with rigidity of the muscles and increased deep and superficial reflexes, says "that such a combination was unlooked for, because in my experience anything like it had never previously been met with. . . . . His condition (as regards the rigidity of his legs and the exaggeration of the deep reflexes) was quite the reverse of what I had hitherto met with in cases where there was complete motor and sensory paralysis in the lower extremities. In such cases, as I have elsewhere pointed out, the limbs are almost invariably flaccid, and the deep reflexes are absent."

Now, when we have the pathological condition of total transverse division of the cord, we have undoubtedly the clinical condition of total motor and sensory paralysis; but the converse does not necessarily follow by any means, and certainly the two statements cannot be used as convertible terms in relation to the loss of the reflexes.

The reviewer (19) brought forward a case of total loss of motion and sensation (of all kinds) in the lower limbs from lateral curvature, but with rigidity of the leg muscles and exaggerated deep reflexes; Thorburn and Bastian both mention similar cases. Gowers and Horsley and Malmotte give cases (already mentioned) where a similar clinical picture presented itself, and due in each case to mere pressure on the cord, but no destruction of it.

It appears, then, that if the cord is totally divided the knee-jerk is lost, but that if only compressed even to the extent of causing total loss of motion and sensation in the legs, the knee-jerk is retained and generally exaggerated.

APPENDIX B.

Since the above review was written a most important paper has come to hand, written by D. Gerhardt, published in the *Deutsch. Zeitschr. f. Nervenheilkunde*, Bd. vi., pp. 127-136, 1894, of which the following is an abstract:—

Gerhardt first gives a short account of some of the investigations on the subject, and points out that if a single case of total division of the cord with reflexes present can be brought forward, it would dispose effectually of Bastian's cerebellar hypothesis. He mentions a case recorded by Barbé (quoted by Sternberg in his work, "Die Sehnenreflexe," Leipzig and Wien, 1893), who examined the reflexes of the body of a man one minute after he had been beheaded, and found them present. Jendrássik (*Deut. Arch. f. klin. Med.*, Bd. lii.) states that many well recorded cases are in opposition to Bastian's theory, but whether these cords were submitted to microscopic examination or not is not definitely stated.

Gerhardt's case was that of a man aged 18 years, who was gradually attacked with motor paralysis of the legs with some loss of sensation. There was total motor paralysis of the legs in three months, and total loss of all forms of sensation below the umbilicus two years after the onset. The muscles of the legs were not atrophied, and the electrical reactions were in the earlier stages, normal. The tendon and skin reflexes were markedly increased, the latter remaining so until death. The urine did not flow away continuously, but was passed involuntarily from time to time; the bowels acted involuntarily when the patient was put into a bath. Bed-sores and a deep abscess near the buttocks were present but healed before death. The upper boundary of the paralysis remained unchanged for four years. The slightest touch on the skin of the legs or of the abdomen caused the legs to be at once drawn up. The legs gradually became more and more contracted, so that in the third year they were constantly drawn up as much as possible to the abdomen; they could, at first, be extended if care was taken, but later on there was great resistance to extension, which was manifestly due, not only to muscular contraction, but also to changes in the joints. In the fourth year the tendon reflexes began to decrease, and in the last six months of life they were entirely absent. The skin reflexes also at this time changed in quality, becoming slower and similar to the contractions of muscles in a state of degeneration when tested by the galvanic current. The faradic and galvanic reactions gradually diminished and finally disappeared, but reaction of degeneration was never obtained. Death took place five years after the commencement of the symptoms, from erysipelas.

On *post-mortem* examination an angioma of the fifth and sixth dorsal vertebræ was found with complete compression of the cord, which for a length of 3·5 cm., was reduced to a band 7·5 mm. broad and 1 mm. thick. In the middle of this area there was absolutely no nerve fibre or nerve cell to be seen, the whole section being composed of arachnoidal tissue. Above the compressed area there was the usual ascending degeneration, and below it the usual descending degeneration. The anterior roots and the ganglion cells of the lumbar region were normal. (It should be mentioned that although those skin reflexes, which caused a contraction of the whole lower extremity, irrespective of the point irritated, were present, yet those which show a more or less defensive movement dependent on the point irritated, such as the abdominal and the cremasteric reflexes, were absent. This fits in with Jendrássik's schema (*Deuts. Arch. f. klin. Med.*, Bd. lii., pp. 5 and 6), who separates the first as pathological spinal, and the second as brain reflexes, and supposes that for the accomplishment of the latter the connection with the brain must be maintained).

Gerhardt points out that the late disappearance of the tendon reflexes in cases such as the above is not mentioned in the text books, but says it is expressly brought forward in Sternberg's monograph (*vide supra*), who states that after marked flexion of the limbs has occurred, nutritive changes take place in the muscles and in joints, almost leading to an ankylosis, and that in this stage the tendon reflexes may diminish. With this opinion Gerhardt agrees, but adds that this decrease can in no way be identified with that seen in Bastian's cases in which the disappearance of the reflexes commenced at once. His case, he concludes, is also quite against the cerebellar theory of Hughlings Jackson and Bastain.

Gerhardt says that the most probable explanation of Bastian's cases is that the arrest of the reflexes is due to an irritation arising from the lesion itself. "We must, from the animal experiments of the Goltz school and from pathological observations in paralyses in cases of cortical epilepsy, which diminish after excision of the scar, regard it as certain that at least a number of cortical paralyses must be considered as due to irritation and not to deficiency. Sternberg postulates a similarity between the interrupting and irritative actions of a lesion, whether occurring in the spinal cord or in the brain, and makes it a reproach to pathology that up to now with a so-called transverse lesion of the brain stem or spinal cord, only the interruption of the current (and here, again, only an interruption of the pyramidal tracts) has been considered. The objection that in the published cases the reflexes have been absent for too long a time for an irritation to still have an effect, is refuted by the analogy with Jacksonian epilepsy; Brown-Séquard (*Arch. de Physiol. norm. et. pathol.*, 1890, ii.) mentions cases in which paralyses, which had been present for twenty months, two years and even (in a case of Horsley's) seven years, had been removed by operation."

This explanation given by Gerhardt is similar to that mentioned by Herter (*vide supra*), and has been already discussed. Even in Gerhardt's own case it is difficult to imagine that at some time during the five years of tumour pressure on the cord there would not be some irritation and a consequent loss of reflexes below, which was, however, not the case.

Although Gerhardt thinks that his case disposes of the cerebellar theory, yet, in fact, this is not necessarily so. For it is possible to imagine that while the tendon reflexes were still present there remained a small portion of grey matter yet unaffected by the scar tissue, and that it was only when it became absolutely destroyed that the tendon reflexes finally disappeared (as they did six months before death).

This case is, then, although a most valuable contribution to our subject, not one which conclusively proves or disproves any special theory.

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Abstracts.

A Case of total Traumatic Lesion of the Spinal Cord at the Junction of the Cervical and Dorsal Regions.

—By Dr. Ludwig Bruns, Hannover. (*Archiv für Psychiatrie*, vol. xxv., pp. 1-72; 2 plates.)

This case offers points of considerable practical and theoretical importance. It is rendered especially valuable by the thorough anatomical examination and full report thereof, containing critical remarks of much interest.

History.—A healthy man, aged 21, suffered, as the immediate result of a fall, dislocation of the spinal column between the bodies of the last cervical and first dorsal vertebræ. The spinal cord at the level of the injury was completely crushed. The patient survived the accident 106 days.

Clinical condition one month after the accident.—Complete paralysis of the lower limbs with absolute flaccidity of their muscles. Paralysis of the trunk muscles. In the arms the motor disturbance was confined to paralysis of the fingers except for extension at their metacarpo-phalangeal joints. The paralysis was symmetrical. In the long flexors and the intrinsic muscles of the hands atrophy and the reaction of degeneration, somewhat more severe in the right than in the left limb.

Knee-jerks absent each side, as also Achilles, vasti and other tendon phenomena.

Sensation of all kinds absent from below the second intercostal space in front, and from below the scapular spine behind. At the second intercostal space itself hyperæsthesia, and down the arms.

Urine dribbles away; the bladder is never emptied without catheterisation. Hard fæces are evacuated involuntarily each third or fourth day. The penis is turgid, and has since the accident been in a condition of semi-erection, which sometimes becomes complete erection on the passage of the catheter.

The pupils are small, the palpebral fissures narrow. There is œdema of both legs. Decubitus. The skin of the legs is cool, very dry, and scurfy. Fever of remittent type.

Three weeks later, i.e., seven weeks from date of injury, the patient's condition had become somewhat different. The paralysis of the lower limbs and trunk remained the same and characterised by complete flaccidity of the muscles. The motility of the fingers had considerably improved, the interossei improving more than the long flexors. Abduction and adduction of the fingers returned almost completely, but flexion of the terminal phalanges remained very feeble; the atrophy of the interossei remained marked; they responded smartly to the constant current. Movement at shoulder and elbow appeared perfect, so also extension at wrist, but flexion at wrist was impaired.

The limit of the anæsthesia had retreated downwards, so that the patient derived definite sensations from skin as low as the level of the fifth intercostal space in front, and the level of the spine of the fifth dorsal vertebra behind.

No return of the knee-jerks, or other tendon phenomena. Bladder, rectum, and penis continued to be in the condition described above.

The pupil of the left eye and the palpebral fissure of the right eye had become somewhat the wider.

Without further alteration of symptoms except those due to progressive general weakness the patient died from exhaustion on the 106th day of illness.

Autopsy.—At the lower edge of the body of the seventh cervical vertebra the spinal cord was found compressed into a flattened band. The rootlets of the eighth cervical and first thoracic nerves were to be seen issuing as grey threads from the compressed portion of the cord.

By microscopical search conducted on the hardened tissues in Weigert-stained sections, &c., it was discovered that the damage to the cord had been most complete at the junction of the eighth cervical and first thoracic segments.

The cord there, as viewed in transverse section, was reduced practically to a mass of fibrous tissue enclosed in thickened but recognisable pial membrane.

The fibrous mass representing the cord contained in it when examined by the Weigert stain a number of inky microscopic points, which were resoluble into degenerated nerve fibres when magnified more highly. Here and there, in certain spots, the cross section of an occasional single fibre would appear normal.

No trace of grey matter or of ganglion cells anywhere. The bundles of the anterior spinal root were altered almost beyond recognition, and completely degenerated; the bundles of the posterior spinal root appeared, on the other hand, completely normal. The author sums up: "Altogether the lesion consisted at this level of a complete destruction of the cord throughout its transverse extent."

Traced upwards the lesion at the top of the eighth cervical segment still amounted practically to complete transverse destruction. By the level of the middle of the seventh cervical segment the grey matter became normal in appearance except that the cells in the ventral horns took a poor stain, and seemed sparse in number, and the rootlets of the anterior spinal root seemed smaller than normal. At this level very little of the lesion appeared to be due to direct trauma, and the ascending secondary degenerations appeared fairly uncomplicated. At the top of the seventh segment there was no trace of trauma.

Traced downwards, the appearances in the upper half of the first thoracic segment were practically the same as in the lower half of the eighth cervical; the anterior roots quite destroyed, the posterior sound. The alterations in the second thoracic segment were evidently in part due to direct trauma, but not below that segment. The lowest trace of the trauma followed the edge of the antero-lateral column of each side. Below that the descending secondary degeneration was seen quite uncomplicated by traumatic extension.

The region of trauma extended, therefore, at utmost from the middle of the seventh cervical segment to the lower part of the second thoracic. Little cavities of necrotic origin, often richly vascular or containing fluid, lay scattered in the region of traumatic sclerosis and its immediates confines. In left posterior column a small cavity in the sixth cervical segment.

Anterior roots, destroyed in eighth cervical and first thoracic segments, severely damaged in second thoracic and seventh cervical segments. Posterior roots of the seventh and eighth cervical and of the first thoracic not injured in their extra spinal course, but posterior roots of second thoracic severely damaged.

Secondary Degenerations.—Upwards: gradual diminution of degeneration until it is confined to the median posterior and a small part of the external posterior column, to the ascending antero-lateral of Gowers and to the direct cerebellar tracts.

Downwards: In the posterior columns two symmetrical patches of sclerosis extending down to the fifth thoracic segment in a position somewhat lateral to and parallel with the lateral edge of the median posterior column. The crossed pyramidal tracts and two symmetrical crescentic antero-lateral areas of degeneration are traceable even into the sacral region. The cord, except in the tracts of degeneration, appears in every way normal, with the possible exception of the cells of Clarke's column.

The author concludes that the posterior roots of the upper thoracic spinal nerves must contribute to the formation of Goll's column, although Marie, from previous evidence, has concluded that they do not. He also discusses the meaning of a pair of sharply defined spots of degeneration in the dorsal angle of the two median posterior columns at the lower end of the second thoracic segment. He points out that Tooth has figured similar patches in a case of lesion at the same level. Author concludes that they are probably definite tract degenerations.

The author points out that if the clinical condition is compared with the best accessible data (Ross, Thorburn), the site of lesion of the motor parts would be expected to extend into the first thoracic segment, while the lesion of the sensory path would be as low as the fifth thoracic segment. The condition found *post-mortem* accorded much much more nearly with the data known for the motor localisation than with that known for the sensory. He concludes, however, that the motor nuclei for the interossei of the hand must extend upwards into the seventh cervical segment. He further points out that the circumstance that the spinal centres for muscles extend over several segments, must render more difficult and uncertain the localisation of spinal injuries by means of the paralyses resultant, and that the diagnosis will have to rest upon careful comparison of the individual paralyses and pareses. He insists on the resemblance of the relation between motor and sensory symptoms in his case to the relation which Thorburn has pointed out to exist usually in cases of injury to cauda equina, namely, that the motor disturbance tends to occupy a higher segmental level in the cord than does the disturbance of sensation.

The author points out very rightly that the condition of the pupils and palpebral openings in this case indicates that Klumpke's conclusion (from clinical data) that the eye fibres of the sympathetic left the cord entirely in the ramus communicans of the first thoracic nerve is too restricted, and that some of the fibres probably run by the second and third roots as well (a conclusion in harmony with experimental observations obtained in the cat and monkey, which render it probable that the main course of the eye fibres is *via* the second root).

The author considers that the case supports very strongly the view of Bastian and Bowlby as to the condition of the tendon phenomena and skin reflexes in total transverse lesions of the thoracic or cervical regions of the cord. A depression of reflex activity for 106 days is of too long persistence to be explained as the effect of shock. He points out that the absence of knee-jerks is all the more remarkable in the presence of the advanced pyramidal degeneration demonstrated to exist in this case throughout the lumbar region. There was, it is true, a slight degenerative change found in some of the muscles and nerve trunks of the lower limb (the nerve to the vastus internus and crureus, and those muscles themselves do not appear to have been selected for examination), but the roots and cord were absolutely healthy, except for secondary degenerations in the latter. The condition of the muscles and peripheral nerves certainly could not account for the absence of the knee-jerk. The case affords positive proof of lasting paralysis and flaccidity of the muscles of the lower limbs, with loss of knee-jerks and skin reflexes, and permanent paralysis of bladder and rectum, in result of total transverse lesion of the cord at junction of its cervical and thoracic regions.

The anatomical report is illustrated by excellent coloured figures of microscopical preparations made from the cord at the levels most important for the investigation of the case.

C. S. SHERRINGTON, M.D.

Spinal and Neural Lesions and Knee-jerk in Diabetes Mellitus.

Minor[1] described alterations in the posterior spinal columns of a youth who had died of diabetes, consisting of a lighter hue of certain tracts in those columns (after hardening in potassic bichromate), which were the seat of slight degeneration, together with some fatty bodies, chiefly found along the walls of the vessels.

Sandmeyer also reported slight alterations in the posterior columns of the cord in diabetes mellitus; and so did Leyden.

R. T. Williamson[2] more recently published two such cases. One, aged 52, had markedly atheromatous arteries, especially those supplying certain wasted and parecified muscles, the nerves

[1] *Archives de Neurologie*, t. xvii., p. 391.

[2] *British Medical Journal*, February 24, 1894, p. 398.

to which latter, on the contrary, appeared normal. There were numbness and tingling in the fingers; loss of knee-jerks, which, however, returned but were not made note of during the last two months of life. No ataxy in walking was observed, and no note was made of symptoms of tabes.

The fresh cord looked normal to the naked eye, but when hardened in Muller's fluid parts of the posterior columns looked pale. In the dorsal region, this change affected different portions and extents of Goll's and of Burdach's columns (but chiefly the former) at different levels. In the lower cervical region, it affected the whole of Goll's columns, and them only; and in the highest cervical region, the posterior two-thirds of these columns. Miscroscopically, the changes in these columns were only slight, consisting of "slight excess of neuroglia connective tissue"; swelling of many nerve fibres; reduced size of outer part of myelin; swelling of many axis-cylinders; and by Marchi's method of staining, scattered degenerated (blackened) fibres in both posterior median and external columns.

In the other case, with loss of knee-jerks, a similar change affected the upper lumbar region extensively, implicating the middle portions, and most of both median and external posterior columns; in the upper dorsal region, the whole postero-external columns alone; in the upper cervical region the posterior half of the postero-median columns alone.

These changes are attributed to the diabetic toxæmia.

Nonne, Eichhorst, and Grube found degenerative changes, or neuritis, of peripheral nerves, occasionally, in diabetes, and with loss of knee-jerk. As to the knee-jerk, taken collectively, with all their individual differences, the cases reported by various observers in which the knee-jerk was lost in diabetes amount, *on the average*, to some 35 to 40 per cent. of the whole. Some look on this symptom, when occurring in diabetes, as of grave prognostic significance; others make little of it in this regard.

Dr. Karl Grube[1] recently published his experience on the knee-jerk in diabetes, adducing 131 cases as being sufficiently definite for this purpose. Of these, the jerk was normal in 113, increased in five, and absent in thirteen. Of the five in which it was increased, the condition in three was said to have been that not of true diabetes, but of glycosuria in neurasthenics, and the increase of jerk attributable to their general neurosis. Of the thirteen in which the jerk was absent, the diabetes was severe in four, and of these one had profound neuritis. In the remaining nine the diabetes was slight, but of these two had tabes, one was so obese that the absence of knee-jerk might have been due to this, and in one slight knee-jerk existed on one side. Thus, of the 131 there were only ten (perhaps only eight or nine) examples of uncomplicated diabetes with loss of knee-jerk.

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[1] Abs. in *Lancet*. March 17, 1894, p. 689, from *Neurolog. Centralblatt.*

With reference to these cases and statistics of Grube, the *Lancet* very rightly suggests that they may largely refer, in reality, to examples of temporary glycosuria, especially occurring in the gouty, and not to true progressive diabetes mellitus; and that neuritis may possibly explain the loss of knee-jerk when it occurs.

W. JULIUS MICKLE.

Reusselaer on the Pathology of the Caisson Disease.
New York Medical Record, August, 1891.

This paper, based on a history of two cases of the disease, and a review of twenty-seven autopsies, with a full bibliography, constitutes the "Merritt E. Cash Prize Essay" of the New York State Medical Society, 1891.

Including, as it does, a very full and carefully reasoned discussion of all the theories of this disease, it not only throws great light on an obscure subject, but enables anyone who will read it carefully to get a good working grasp of its pathology, and to explain easily and completely nearly every symptom it presents.

In the two cases first mentioned, symptoms of paraplegia manifested themselves in thirty minutes and in five minutes respectively, after the locking out of the Caisson was completed.

In Case 1, with a good family history, and absence of syphilis and alcohol, the trouble began with a slight pain in the abdomen: he began to improve at the end of a week, and in six months could again walk quite well.

In Case 2, there was a marked family history of phthisis, and to this the patient had added alcoholism and syphilis, the latter being contracted three years before the Caisson disease.

The paraplegia in this case was preceded by giddiness, weakness and pain in the head and legs; he got steadily worse, developed a gangrenous bed-sore, and died in about five weeks.

On examination of the cord after hardening, marked pathological changes were found in the dorsal region over an area of two inches. These affected specially the posterior and adjacent lateral columns, but the grey matter was normal. The microscope showed destruction of nerve tissue with degeneration or absence of axis-cylinders, and increase of neuroglia at the focus of the lesion, with ascending and descending degeneration above and below it.

The first twenty-five autopsies, which are defective both in examination and in reporting, show, so far as they go, congestion or softening of the cord; congestion of the brain and of most of the internal organs.

Then follow two cases in which a microscopical examination of the cord was made, and this shows, much as in the author's own case, a diffuse parenchymatous myelitis with its focus in the lower dorsal region, to which the author concludes that Caisson disease is due.

For the purpose of discussing the theories of causation he groups them under three heads.

(1) The theory of exhaustion and cold.

(2) The gaseous theory.

(3) The theory of congestion with sequelæ, which are: (*a*) Black blood. (*b*) Evolution of gas in blood vessels. (*c*) Hæmorrhage. (*d*) Acute revulsive anæmia. (*e*) Comparative stasis.

(1) Attributes the pains to rheumatism, and the paralysis to reaction after exhaustion, and not to inflammation. Yet congestion, effusion and inflammation are found *post-mortem*, and again, exposure to cold and exhaustion, as in winter, does not produce similar symptoms.

(2) Which is concerned specially with the idea that gas is set free in the blood on the withdrawal of pressure, is the theory which the author specially attacks, while, as we shall see, he gives his adhesion to the theory of congestion with the sequelæ of comparative stasis (3 *e*).

This theory (2) has many supporters, and the experiments of Paul Bert are quoted to prove the evolution of gas in the vessels; but the author points out that Bert himself shows that it requires a pressure of at least five atmospheres followed by withdrawal of pressure within three minutes, to produce bubbles of gas in the vessels, while men are practically never subjected to so great a pressure, or to such rapid withdrawal, so that gas cannot be the cause of their symptoms.

The author further quotes the experiments of A. H. Smith, to show that the amount of gas that can be evolved from the blood is really very small, while he points out that a considerable quantity of air can be slowly introduced into the vessels without effect.

He also points out that the advocates of the gaseous theory acknowledge that length of exposure to pressure increases the danger, while physical laws make it probable that the blood absorbs gas as quickly under increasing pressure as it gives it out under diminishing pressure ; so that length of exposure to pressure beyond a few minutes does not increase the amount of gas absorbed, or the amount that will be given out on removal of pressure, and the gaseous theory thus fails to explain the increased danger of lengthened exposure to pressure.

Again, it has been shown that birds and amphibia can stand much higher pressures than mammalia without inconvenience; but the physical laws which govern the saturation of the blood with gas apply equally to all these classes of animals, and we shall see presently that the theory of congestion followed by stasis suffices to explain these points where the gaseous theory fails.

The theory of congestion followed by black deoxygenated blood is founded on the supposition that the congestion and deficient circulation in all the internal organs during the exposure to pressure does no harm, because there is an excess of oxygen in the blood ; but when the pressure is removed the blood becomes poor in oxygen, and then the congestion does harm, and there is an accumulation of effete matters.

As a matter of fact, under the pressures in common use there is but little alteration of the oxygen in the blood, and as the author further points out, the accumulation of effete matters would be too slow to account for the prompt effects often seen.

The theory of congestion followed by the evolution of gas is to a large extent open to the objection urged against the gaseous theory alone.

The theory of congestion followed by hæmorrhage is disposed of with the assertion that the hæmorrhages found have never been large enough to press on the cord and cause paralysis.

As to the theory of congestion followed by acute revulsive anæmia, the author quotes Moxon, to the effect that the brain can withstand very great pressures if it is not removed too quickly, and also as to the poor blood supply and natural difficulties affecting the circulation of the cord and cauda equina.

Also Gowers, as agreeing with Moxon that the lesion is due to a revulsive anæmia, *i.e.*, first a congestion due to the pressure driving blood from the surface of the body followed by anæmia as the conditions are reversed.

And Twynam, as further suggesting that the increase of pressure drives some cerebro-spinal fluid out of the cranium into the spinal canal, and that this, by pressure, produces anæmia of the cord.

The author, however, disposes of all these suggestions on the ground that the cord is not found to be anæmic, but almost invariably congested.

We now come to the theory which the author supports, that of congestion followed by comparative stasis, and he quotes Smith and Nixon as showing that the vessels of the deep organs are first paralysed by continued over-distension, and then, as the pressure is removed, there is a general loss of arterial vis a tergo, so that stasis supervenes.

Also Professor Birmingham as to the special difficulties of the arterial circulation in the lower part of the cord, which tend to favour the occurrence of stasis then under the above conditions.

And some experiments of C. A. Herter, which show that occlusion of the abdominal aorta and vena cava at once produces paralysis of the lower extremities.

Finally, the author points out that the two things which slow the blood current are (1) paralysis of the vessels in deep-seated organs by continued over-distension, the paralysis being greater the longer the exposure to the high pressure; and (2) the general fall of blood pressure which ensues when, with the removal of pressure, the vessels in superficial parts are again freely open to the blood stream.

The result to the deep organs is stasis, perhaps thrombosis, with correspondingly deficient nutrition.

In favour of this he adduces the increase of urine which takes place under the plus pressure as evidence of increased blood pressure in the kidneys, as well as of diminished functional activity in the skin.

The evil effects of alcoholism and debility he explains by reference to these powers of diminishing the resistance of the vessels to over-distension.

Of obesity by the fact that the obese have more blood than the thin, and hence the congestion of their deeper organs will be greater.

While the protection of repeated exposure is said to be due to the vessels gradually acquiring power (as is also the case in birds and amphibia) to withstand the plus pressure.

Increased duration of pressure increases as we have seen the congestion and paralysis of vessels, and exposure to cold and moisture causes general debility and fatigue and weakens the vasomotor system; while, on the other hand, the gaseous theory quite fails to explain any of these points.

The evil action of heart disease, kidney disease, lowered vitality, or old age is quite obvious; while the anatomy and physiology of the circulation of the dorsal cord explain the frequency with which it is affected.

A. Haig.

BRAIN.

PARTS II. and III., 1895.

Original Articles and Clinical Cases.

BRAIN ORIGIN.[1]

BY SIR WILLIAM BROADBENT.

The honour of presiding over this Society is one of which I am peculiarly sensible, and I am grateful to the Council and Members for bearing in mind that at one period of my career I was more or less a neurologist, though the name was not then invented. I was, in fact, one of a band of men enthusiastically engaged in the study of the nervous system and the diseases to which it is liable, and my oldest and best friends are still among those with whom I thus became associated.

I might well take as the subject of my address the stupendous progress realised in our knowledge of the physiology and pathology of the nervous system since that time. Lockhart Clarke was only then giving us a definite idea of the structure of the spinal cord and bulb, and Brown-Séquard was publishing his brilliant researches on the function of its different columns and strands of fibres, and on the motor and sensory paths. The ophthalmoscope, Helmholtz's splendid invention, had not then been applied to the study of diseases of the brain. The two last names, both immortal in the annals of science, I bring together because they have been erased by death from the list of corresponding members of this society during the past year. At that time Charcot, whose loss we had to deplore in the previous year, had only just entered upon the extensive field of work afforded by the Salpêtrière, and his gigantic contributions to neurology were still in the womb of the future. Virchow's teachings with regard to embolism and its results had not been applied to cerebral pathology, and our teachers of that day spoke of red or inflammatory softening. The significance of the knee-jerk was unknown. The localisation of cerebral functions was scarcely even dreamt of: Broca's identification of the third left frontal convolution as the seat of articulate speech, the first instance of the kind, had, it is true, been established for some time as an isolated fact. Then as Dr. Hughlings Jackson was pointing the way by a new interpretation of clinical observations, Hitzig and Ferrier stepped in, and, the latter especially, by a series of experimental investigations, as profound in conception and as fruitful in result as any in the history of physiology, mapped out the cortex according to the functions of the different convolutions.

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[1] Being the Presidential Address to the Neurological Society of London, January, 1895.

In the short time during which I shall occupy the attention of the Society, however, I shall not attempt any such comprehensive review of the progress in our knowledge of diseases of the nervous system or an exposition of any special form of nerve disorder. Still less shall I emulate former Presidents of this Society who have made of their introductory addresses important contributions to our knowledge of the anatomical structure, or of the physiology of the nervous system. I should only lay myself open to criticism from the youngest member here present, but I am encouraged by the example of Dr. Gowers to carry out the idea which suggested itself to me as soon as I learnt that the responsibility of addressing you had devolved upon me. It is to bring to your notice certain speculations as to the nature of nerve force and as to the constitution and mode of action of the nervous system which have long occupied my mind.

Dr. Gowers, in his admirable address before the Medical Society of Manchester, has rendered me a double service. He has vindicated in eloquent and convincing language the utility of speculation and hypothesis as inspiring and directing thought, and suggesting and guiding experiment, thus clearing my way and making any such effort on my part unnecessary, and again by his lucid and exhaustive discussion of the dynamics of life, he has spared you and me much detailed exposition, which in my hands might have been tedious. He has given increased vividness and precision to the notion of nerve force as a mode of motion correlated with the more familiar modes of motion, heat, light, electricity, chemical action, and mechanical energy, and also to our ideas as to the nature of the various stimuli which excite nerve action, pointing out that, like the concussion which explodes nitro-glycerine, or the heat which fires gunpowder, or the electric spark which determines the sudden combination of a mixture of oxygen and hydrogen, they are essentially added motion.

Nerve action, whatever the difficulties of giving a chemical explanation, can scarcely be other than motion liberated by molecular change, in other words by chemical change in the highly specialised nervous structures.

We cannot so clearly as in the case of muscular action show that the development of nerve force is attended with an increased output of CO_2 and water and obtain a quantitative evaluation of it by the amount of oxidation required for its production, but I think we may say that we have absolute proof that nerve force is liberated by a process which for want of a better term we must call chemical in the dependence of the functional activity of the great nerve centres upon a due supply of oxygen. This is best shown by the successive suspension from above downwards of the functions of the cerebral cortex, basal ganglia, and of the vital bulbar reflexes when the supply of the oxygen is cut off, or when the oxygen of the hæmoglobin is replaced by the anæsthetic gases or vapours. From the fact that oxygen so evidently takes part in the process, it would almost seem as if the nerve motion were liberated by atomic rather than molecular changes, though this is not a necessary conclusion.

There are many considerations which appear to indicate that nerve force must be characterised by intensity rather than by quantity—that, for example, it would compare in quantivalence of motion with static or faradic electricity rather than with galvanism. A single cell with an adequate coil and core can yield faradic currents of intolerable strength, and it has been said that the electric force required to dissociate the hydrogen and oxygen of a grain of water would make a very respectable thunderstorm.

Among these considerations is the fact already alluded to, that it has never been possible to distinguish and measure the products of the oxidation by means of which nerve energy is liberated. Another is the remarkable promptitude of nervous operations of every kind, and the accurate adjustment of response to stimulus. Yet another is the marvellously minute dose of drugs acting upon the nervous system which is sufficient to produce striking effects. If the correlation of forces is to apply to the physiological effects of drugs—which is an article of scientific faith—then the chemical change in the morphia molecule must be equal and opposite to the change in the constituent molecules of the cerebral cortex which the morphia neutralises when it suspends the cortical functions.

Now the chemical correlative of intensity as the characteristic of nervous energy is the existence in the nerve centres of a substance possessing high chemical tension. By this is meant, not simple instability, not necessarily instability at all, but a molecular structure in which the atoms are grouped in a way which is not in accordance with what may be called their natural affinities, so that if once free to move they would rearrange themselves more or less abruptly and perhaps violently in a totally different manner.

Instability may be described as a readiness to yield to the disintegrating influences of forces acting from without; chemical tension implies the existence of an intrinsic tendency which under special conditions determines change in a definite direction and with a certain degree of energy. Nitro-glycerine, for example, or gun-cotton does not undergo gradual decomposition, and the former even resists the application of flame, while concussion determines in both a re-arrangement of the constituent elements attended with violent explosion.

Now nitrogen is the element which lends itself to sudden disruption of a complex molecule. It has very weak affinity for hydrogen and oxygen, none at all for carbon, while carbon, hydrogen and oxygen have powerful affinities for each other. The tension in nitro-glycerine and gun-cotton is brought about by the substitution of NO_2 for hydrogen in the molecule of glycerine or cellulose. Before this the molecule was stable but was ready to burn; *i.e.*, its carbon and hydrogen to leave their combination with each other and combine with oxygen. The introduction of additional oxygen in the NO_2 upon which the associated nitrogen has only a feeble hold may be compared to the mixing of oxygen with coal gas.

The chemical tension by means of which nerve force is evolved would not be of the same character as that exemplified by nitro-glycerine but would more probably resemble that produced by the forced departure from a stable molecular type, as, for example, by the successive removal of hydrogen from the ammonium molecule. Amides, imides and nitrites are formed as one, two and three equivalents of hydrogen are abstracted, in which the affinity of nitrogen for hydrogen, which is satisfied in the ammonium molecule, is left increasingly unsatisfied, each step in the process, generating an increasing tendency to return to the natural molecular structure. It is this inherent tendency towards a normal balance of affinities and forces which for want of a better term we call chemical tension.

When we contemplate nervous force from a chemical point of view an obvious conjecture is that, whereas most forms of nerve action are attended with chemical change in the direction of oxidation and disintegration, the impinging of sensory impressions on the cerebral cortex, not giving rise to immediate response of any kind but registered as memory, may be attended with change of a converse character, integration of molecules and structure by means of which the mental picture is rendered more or less permanent. Another reflection which forces itself upon the mind is that, when the energy liberated in all the various forms of nervous activity has been accounted for by chemical changes in the nervous structures concerned, and nerve force has been correlated with other modes of motion, we are left with sensation, consciousness and the higher mental operations as surplus. We cannot bring them into the reckoning at all; the phenomena of mind are outside and above and in excess of the material changes which subserve their manifestation.

Turning now to a speculation of another kind, I think I am not misrepresenting the prevailing idea as to the nature of nerve force, and as to the relation between the nerve centres themselves and between them and the periphery, when I say that nerve force is considered to be energy which is transmitted from point to point of the nervo-muscular apparatus; that, for example, in the production of muscular action a certain force is supposed to be generated in the centre and transmitted to the muscles upon which it acts as a stimulus, the muscles awaiting passively the added motion in response to which they develop mechanical energy. This idea of the origination and transmission of energy pervades our entire conception of nervous operations, a centre or end organ being looked upon as the passive recipient of an impulse or force which is evolved in the centre in which the energy is liberated and from which the impulse starts.

Now it has long seemed to me that a conception which better fits in with all the phenomena of nervous action is, that instead of this passive attitude of the periphery and centres one to the other and of the centres, sensory and motor alike, towards each other, the relation in all cases is one of mutual tension or resistance—that is, that at all points specialised for the liberation of nerve motion, latent energy is generated and exists in a potential form, which, in the absence of restraining conditions would become active, and that the restraining condition is a counter-balancing energy developed in the centres or peripheral nerve expansions with which any given point is connected by nerve fibres.

In a galvanic battery, which may be taken as an illustration, the powerful affinities of sulphuric acid and zinc are held in check so long as there is no connection between the carbon and zinc elements; and a state of tension or charge exists in the wires carried from the carbon and zinc respectively which, when the number of cells is sufficiently great, is betrayed by the leap of a spark from one electrode to the other, or by the passage of a current through water which it decomposes on its way.

The telegraph wires, again, in the intervals during which no messages are passing are not simply in a passive state; they are charged with electricity by the batteries at the two ends which are held in an equilibrium of mutual resistance through the charge which they communicate to the wires, so that disturbance at one end in a plus or minus direction gives instant rise to a disturbance at the other, corresponding exactly both in direction and in degree. The telegraphic instruments are, so to speak, in touch with each other. The same may be said of the nerve centres and peripheral nerve endings; they feel each other by the exercise of a sort of opposing pressure from each end through the connecting nerves, like two fencers whose swords are crossed.

Without saying that this conception removes the difficulty in understanding the marvellous correspondence of sensations with impressions, or the equally marvellous adjustment of muscular movement to motor nerve impulses, it may perhaps bring it nearer to our comprehension.

All who have attempted to grasp the mystery of nervous action have recognised that the nerve endings are highly specialised and endowed with some powers allied to those of the centres. This is especially evident in the case of the sensory nerves, and attains its highest development in the retina and auditory nerve. The termini of the cutaneous nerves must equally be specialised though the apparatus is less conspicuous, and, just as it is not light or sound which is transmitted to the brain, but nerve motion set up in the retina or auditory expansions by luminous vibrations or sonorous pulsations, so nerve motion is excited in the nerve endings of the skin by impressions of various kinds. In the eye and ear it is seen that structures are employed similar to those found in the nerve centres, having presumably therefore similar endowments, and in particular the power of generating nerve energy, and although nerve cells are not recognisable in the skin, the specialised nerve endings must possess a power corresponding to that of the retinal expansion. There can be no difficulty, then, in supposing that at the sensory terminal distribution generally, force is continuously generated and maintained at a certain degree of tension, which is balanced and resisted in the nerve centres by force there generated.

In the same way the relation between the motor nerve endings and the motor nerve nuclei in the cord would be one of balanced tension. The muscular tonus which gives rise to the knee jerk would appear to be the peripheral expression of such a relation.

The various centres again in functional association with each other will stand in this relation of mutual stress, each generating force which exactly balances that of the associated centre or centres during repose, and the one responding to the slightest change set up by a stimulus in the other.

We may compare this relation of different nerve centres to two groups of men engaged in a tug of war. There is no movement in one direction or another so long as the two sides are exactly balanced in strength, but let one man slip, or become exhausted, or another gain a better foothold, and there is at once a movement in one direction or another of every member of his own group, which is accompanied by an exactly corresponding movement in the other. Another illustration may be suggested as perhaps illustrating the relation between a centre and a nerve end—a small group of men at the long arm of a lever resisting or compelling movement of a much larger group at the short end.

This relation of mutual tension and reciprocal control covers the whole field of operations governed by the nervous system. No separate mechanism of centres and nerves is required for thermal, trophic and inhibitory operations.

The centres and nerves which subserve sensation and motion are also employed in the liberation and control of heat. The thermal centres supposed to be identified and localised by experiment practically coincide with and are the same as the motor centres. As regards the part taken by the nervous system in the production and regulation of the body heat, it would appear from the occurrence of hyperpyrexia after fatal injuries to the brain, and in the course of certain diseases simultaneously with extreme nervous prostration, and from the rise of temperature sometimes observed after sudden death or very rapidly fatal disease, that the tendency in the blood and tissues is to oxidation and excessive heat production, this being held in control by the nervous system. The higher temperature found in the paralysed side in the hemiplegia points to the same conclusion. It seems probable that pyrexia may result sometimes from acceleration of oxidation in the structures overcoming the central thermotaxic control, at others from impairment of the control exercised by the nervous system.

In the same way this conception of the relation between the periphery and centres and between different centres goes far to explain inhibition. The normal relation between all parts of the nervous system would, in fact, on this hypothesis be one of mutual inhibition or control, and it would follow that a powerful impression on one centre, in other words, an unusual development of energy, at a particular point, might at the same time excite increased activity of one set of associated centres, and resist and annul increased motion in another centre or set of centres.

One of the most remarkable examples of inhibition is seen in the prevention of an attack of Jacksonian epilepsy by tightening a ligature round the limb which is the seat of the initial convulsion. A coarse lesion exists in some part of the motor cortical area; irritation dating from this lesion sets up from time to time convulsive movements in the limb whose motor centre is the seat of the disease, and yet although there is irritation in the motor centre itself, the convulsion can not unfrequently be arrested by a tight cord round the limb. A fact like this can scarcely be understood except on the hypothesis that the periphery and centres hold each other in mutual control.

So with regard to trophic influence. The nerve endings in skin and muscles are identified in their nutrition with the structures to which they are distributed. The muscles, indeed, may almost be looked upon as end-organs of the motor nerves. Through this community of structure and nutrition with the nerve terminations it seems to me that the tissues will take part in the maintenance of the tension between periphery and centres, while in turn the nerves will influence the nutritional processes in the tissues just as, by way of analogy, heat and electricity condition chemical action.

We see nutrition of the tissues disturbed by influences transmitted both along motor and sensory nerves with or against the direction of the physiological nerve impulses. The results are more striking in the latter case, as is exemplified by acute bed-sores, herpes and ulcerations, all of which are excited by disturbing influences transmitted from the cord along the sensory nerves to the skin.

It is remarkable, again, that in herpes the most common if not the most interesting illustration of trophic lesions, the vesicles come out in scattered patches so that only a small part of the area of skin, to which the nerves conveying the disturbing influence are distributed, is affected. In some cases of frontal herpes, however, the skin of the entire region may subsequently undergo atrophy, and be left thin and glistening; and after a severe blow on the side I have seen two intercostal spaces crowded with vesicles from the seat of the injury to near the edge of the sternum. Perhaps Dr. Head, who has made such excellent use of herpes in his instructive researches with regard to cutaneous tenderness in visceral disease, can tell us whether the groups of vesicles occupy approximately the same situation in different cases. A certain degree of constancy is suggested by the patch which always appears on the back in zoster.

Herpes is often attributed to irritation or inflammation of a nerve, but it appears to be the result of a central disturbance, probably reflex, and not in any true sense of a neuritis. Labial herpes is curiously associated with the general commotion of the nervous system, of which rigor is the expression, and zoster in all its varieties is probably a visceral reflex. As Dr. Head has pointed out, the distribution of the patches is in relation with a segment of the cord, and not with the termination of a given nerve or nerves. The neuritis which undoubtedly exists and keeps up the pain may be secondary to the central explosion, and, like the herpes, due to it.

Another point, of no importance in itself, but worthy perhaps of a few minutes' thought and attention, is the localisation of tactual impressions. From the earliest days of experimental investigation it has been recognised that division of a sensory nerve did not extinguish sensation in the skin to which it is distributed in the same definite way that division of a motor nerve paralyses muscles, and it is found that there is overlapping of sensory areas. This appears to prove conclusively that the localisation of an impression on the surface is not due to conduction of the nerve motion along an individual fibril starting from that particular point. Every papilla of the skin would, on this supposition, need to have two or more nerve fibrillæ, and it is already difficult to imagine that the cutaneous nerves can contain a sufficient number of fibrils to supply every point on the surface, however finely the axis bands may be sub-divided. The localisation of a sensation, it seems to me, must be effected by a sort of triangulation. In land surveying the exact position of a distant point is fixed by measuring the angles at the base of a triangle of which it forms the apex. So an impression made on a given spot excites nerve motion which is conveyed to the spinal cord by two or more nerve filaments approaching the spot from different directions, and its precise localisation is arrived at by continuous experience based upon the relative intensity of the motion set up in these converging nerve filaments. Impressions on adjacent parts will send currents a little more intense along one channel, a little less along another; and it must be by combination and comparison of the motion reaching the cord by these different channels that the point of origin is recognised. The entire papillary layer of the skin is, in my apprehension, a sentient expansion—a tactile retina, tapped at points more or less close by the sensory nerves.

A further difficulty in accepting the idea that the localisation of a sensory impression is due to transmission along a single infinitely minute fibrilla proceeding directly from the point of origin to the cord, arises when we consider what happens when a sensory nerve is re-united after division. Nothing could be more improbable than that the coaptation should be so exact that every one of these fibrils should meet so that the continuity of each should be restored, without which sensory impressions would be wrongly placed. On the supposition under consideration the accurate localisation of sensations would not depend on the continuity of individual fibrils, but would be re-acquired by experience *pari passu* with the re-opening of the paths for centripetal impressions.

The reunion of a mixed nerve opens up further questions. It must happen that sensory fibres on the proximal side of the section will find themselves end to end with motor fibres on the distal side, and, in the absence of some condition which prevents it, they would unite, to the confusion of the nervous arrangements of the muscles and skin supplied. Apparently, however, this does not take place, and it must be assumed that efferent impulses, on the one hand, and afferent tendencies on the other, determine the establishment of the new communications. This implies some sort of dynamic influence from both proximal and distal ends, which is not easily understood on any other hypothesis than that advanced this evening, namely, that the nerve nuclei in the cord and nerve expansions in the muscle and skin stand in a relation of mutual tension, which tension exists in the nerves.

I will conclude this halting address by a few remarks on the much-debated question of the unity or multiplicity of the conductors of sensory impressions.

An important point which Dr. Gowers brings out is that the conduction of impulses along nerve fibres is effected by molecular change, as well as the evolution of nervous energy in the centres, and by change probably similar in kind, though differing in degree and especially in intensity. This conception appears to me to remove all necessity for postulating the existence of special conductors and centres for tactile, painful and thermal sensations.

The basis for this assumption is the fact that the different forms of sensibility are not necessarily impaired concurrently, but that one may survive when the others are lost or perverted. But the degree or kind of motion making the impression is different in each case, and the motion set up in the conducting fibres and communicated to the sentient centres must be different. A tactile impression has only to be intensified to produce pain, and in certain states of the nervous system, central or peripheral, the lightest touch is intensely painful. Heat, again, and cold—which last, by the way, must subtract motion—carried beyond a certain point cease to be distinguished one from the other, the only sensation being one of pain.

Neither the experiments nor the clinical observations which have been held to demonstrate the separate paths and centres for these different modes of sensibility have at any time seemed to me conclusive; the lesions or morbid changes or drugs supposed to affect certain special fibres and centres may more probably impair the susceptibility of common or general conducting paths or termini to certain modes of motion, or their power of taking on such motion.

A consideration which has always seemed to me inconsistent with the existence of separate paths and centres for sensations of touch, pain and temperature is that they are so commonly lost in the same order, tactile sensibility surviving pain and the appreciation of heat and cold. The loss or impairment of sensibility to pain and variations of temperature, while tactile sensibility remains little affected, is stated to be so constant in syringo-myelitis as to serve as a diagnostic characteristic. Surely the structures of the cord are not affected in so constant an order that the tactile paths are never involved, while the fibres or networks which serve as the channels for pain and sensations of heat or cold are always destroyed.

Such a condition as the following, of which a well-marked example is under observation at the present moment, appears to me to be incapable of explanation by the hypothesis of multiple distinct sensory channels and centres, and to point to modification of a conducting and receptive apparatus common to all the different forms of cutaneous sensibility.

The condition is that tactile sensibility being good and sensitiveness to pain much exalted, there is absolute loss of sensibility to thermal impressions, any departure from an average temperature being felt as pain, whether in the direction of heat or cold, the patient being unable to say which of the two is being applied. The case is one of left hemiplegia, established three years ago during an attack of influenza. There was absolute loss of sensation over the entire left half of the body, and of motor power in the limbs. A considerable degree of movement has been recovered and improvement is still going on; there is no rigidity of the kind common after paralysis, but a certain degree of contraction of the flexor muscles still persists. Sensation slowly returned, pain being a marked feature from an early period.

If we consider the order of disappearance of the different forms of sensibility from the point of view that the peripheral receptive nerve expansions, the afferent fibres, and the central sensory termini are the same for all, this order becomes significant when compared with the order in which sensibility to different kinds of impressions must have been evolved. Tactile sensibility has been looked upon as the most intellectual of the cutaneous sensations, and the inference has been drawn that it was therefore the highest and latest in order of evolution, and consequently, according to the law on which Hughlings Jackson so constantly insists, it should be the first to disappear. But is this inference legitimate?

When we consider the rudimentary forms of life, it is clear that the existence of the creature must have depended upon an accurate response to surface impressions, which implies that such impressions must have been exactly localised, and that their character, *i.e.*, whether nutrient or not, must have been recognised. This is nothing more or less than a rudimentary sense of touch. The sense of pain would correspond to the recognition of impressions as being too violent for the safety of the part of the animal exposed to them. A localised recognition of temperature changes would not be required in the lower forms of animal life leading an aqueous existence. We have thus as much conformity with the law that the order of dissolution corresponds with the order of evolution, as is met with in clinical experience generally.

I trust that these fragmentary speculations may not have been altogether unworthy of the attention of the Society.

POINTS RELATING TO THE WEBER-FECHNER LAW. RETINA; MUSCLE; NERVE.

Being the substance of a communication to the Neurological Society, March 7, 1895.

BY A. D. WALLER, M.D., F.R.S.

THE Weber-Fechner law in its restricted sense designates the relation between magnitude of sensation and magnitude of stimulation. I am using it at present in a more extended sense to designate the relation between cause[1] and effect in living matter, and as instances that I have been able to study I shall bring under your notice experiments upon (1) the retina, (2) muscle, (3) nerve.

The ordinary view of the Weber-Fechner relation between sensation and stimulus presents the former as a logarithmic function of the latter.

Accepting this as in the main correct, and deferring discussion of the departures from the law that take place at the higher and at the lower limits, let us first face this definite question: there is a disproportion between stimulus and sensation such that equal increments of sensation require increasing increments of stimulation, or—what amounts to the same thing—that each equal increment of

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[1] The word "cause" is here used in its popular sense, to denote the immediate discharging cause, not in its strict physical sense, when of necessity cause and effect are equal and opposite. Evidently it is only in the first sense of "discharging cause" that the stimulus of a nerve is the cause of muscular contraction, just as in the same sense a spark is called the cause of an explosion, or a match the cause of a fire. The unstable equilibrium of fuel, or of powder, or of nerve-matter is obviously a factor in the aggregate causation, particular elements of which are treated as terminals of thought when, *e.g.*, we consider the relation between magnitude of stimulus and magnitude of contraction, or between magnitude of stimulus and magnitude of sensation.

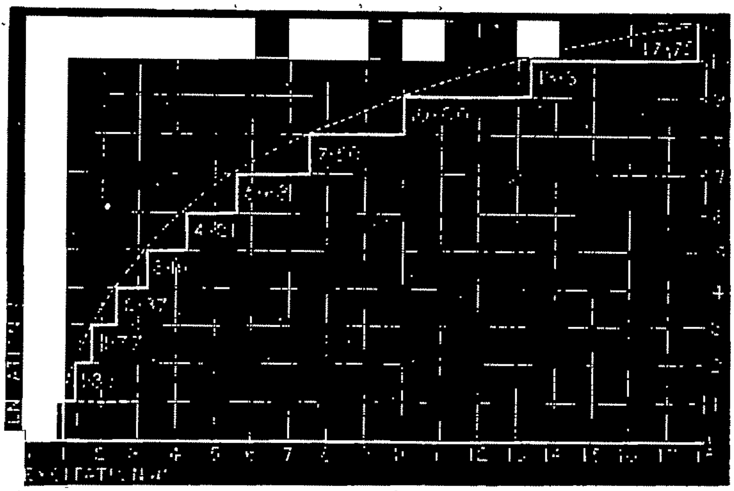

FIG. 1 A.

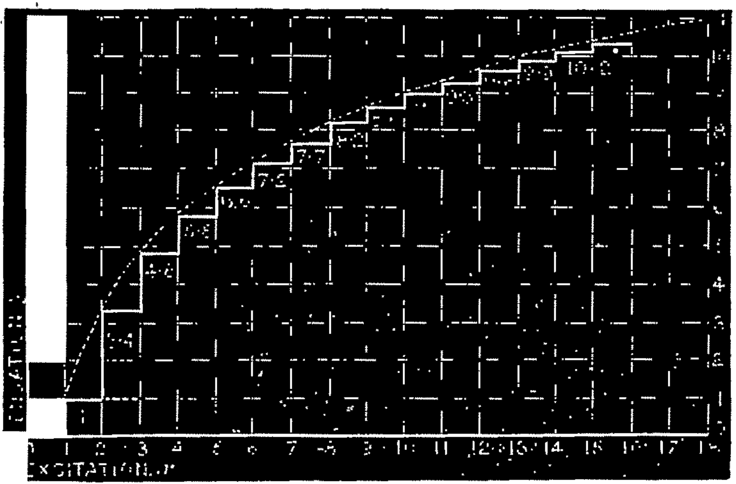

FIG. 1 B.

The Weber-Fechner curve, expressive of a logarithmic relation between excitation (increasing along the abscissa) and sensation (increasing along the ordinates). The two curves, A and B, are identical. In A the curve is obtained by taking along the abscissa the magnitudes of excitation required to produce "the minimum perceptible difference" between successive stimuli assumed by Fechner as the unit of sensation. In B the curve is obtained by calculating the magnitudes of sensation corresponding to an arithmetical increase of excitation. Both curves show, therefore, the same relation, A showing that to equal increments of sensation increasing increments of stimulation are necessary, B that equal increments of stimulation cause diminishing increments of sensation. In A the steps of sensation are equal, but each tread is longer than its predecessor; in B the steps of sensation diminish in the ascending scale, their treads being equal. The curves are constructed from numbers corresponding with an increase of excitation by $\frac{1}{3}$ necessary to produce a perceptible increase of sensation.

stimulation produces a diminishing increment of sensation. Is this disproportion of physiological or of psychological origin?

To present this question clearly it is convenient or even necessary to distinguish two stages in the passage from external physical stimulus to sensation; a not infrequently committed ambiguity in the use of the word "stimulus" will thereby be avoided.

We have :—

(*a*) the external stimulus, *i.e.*, the objective phenomenon or external cause of excitation apart from the excitation itself.

(*b*) the internal stimulus, *i.e.*, the material sensificatory change, provoked in the nervous system by the external stimulus.

(*c*) the sensation itself, *i.e.*, a subjective phenomenon concomitant with the sensificatory change.

Given, *e.g.*,

A series of external stimuli of magnitudes 1, 2, 3, 4, 5, &c., and a series of sensations of magnitudes 1, 3, 5, 6, 7, &c. Does the disproportion take place on physiological ground between external stimulus (*a*) and internal stimulus (*b*), or on psychological ground between internal stimulus (*b*) and sensation (*c*)?

Some contribution towards an answer to this question would be afforded if we could measure and express in any scale of units a series of internal sensificatory changes—effects of a series of external stimuli, their cause.

I.—On these grounds I have bestowed a considerable amount of attention upon the RETINA, where, thanks to the labours of Holmgren, followed by Dewar and MacKendrick, Kühne and Steiner, and others, we know that the stimulus of light produces an electrical change that may be utilised as the objective sign of a physico-chemical change in the retina. It is not proved, but it is highly probable, that such physico-chemical change is the sensificatory phenomenon in so far as the retinal portion of the retino-cerebral organ is concerned. For the purposes of the present inquiry this may be assumed, and we may proceed to measure out quantity of external stimulus (light), and read off upon the galvanometer quantity of physico-chemical change, considering such physico-chemical change as an instance of internal stimulus or sensificatory phenomenon.

Here is an example of retinal action consequent upon luminous stimuli of increasing magnitude.

The magnitude of stimulation was in this case varied by altering the distance from the retina of a standard candle; the object submitted to experiment was the freshly-excised eyeball of a frog, resting upon one electrode by its posterior surface, which included the cut stump of the optic nerve, led off to the galvanometer by that electrode and by a second electrode, the actual end of which consisted in a saline-moistened thread resting upon the edge of the cornea. The eyeball was adjusted inside a dark box opposite to a blackened tube, through which light was let in or blocked off by the movements of a shutter not connected with the box, moved by hand or by an electro-magnet. The cornea was directed upwards—light impinged upon the sclerotic, glancing over and reflected from the surface of the eye-ball, so that there was no appreciable focussing of the candle flame through the refractive media of the eye. In the

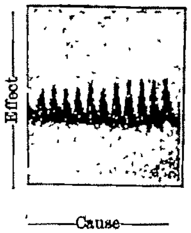

FIG. 2.

Fig. 2.—Galvanometric deflections (*i.e.*, retinal effects) in a frog's eyeball caused by exposures lasting one-eighth of a minute at minute intervals to the light of one candle at the distances 14·1, 10, 7·07, 5·77, 5, 4·47, 4·08, 3.78, 3·53, 3·33, 3·16 feet giving stimuli of the strengths: 0·5, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10 units, the unit taken being equal to $\frac{1}{100}$ of a standard candle at a distance of one foot. The curve obtained by joining the summits of the ordinates closely resembles the Weber-Fechner curve of fig. 1.

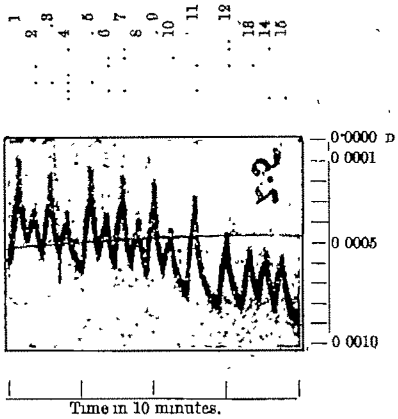

FIG. 3.

Fig. 3.—Galvanometric deflections by retinal currents produced by steady and by interrupted illumination of different intensities.

(1) Steady light at 10 units for 1 min.
(2) Interrupted ,, ,, ,, $\frac{10}{4}$ or 2·5 units.
(3) Steady
(4) Interrupted
(5) Steady
(6) Interrupted
(7) Steady
(8) Interrupted
(9) Steady
(10) Interrupted
(11) Steady at 40 units for 1 min.
(12) Interrupted ,, ,, $\frac{40}{4}$ or 10 units.
(13) Steady at 2·5 units for 1 min.
(14) ,, ,,
(15) ,, ,,

N.B.—The interruptions of light were caused by a revolving disc with open sectors giving 10 interruptions per sec. The sum of the sectors occupied $\frac{1}{4}$ of the whole disc.

The particular points illustrated in this figure are:

(1) That at equal strengths interrupted light produces a smaller effect than continuous light.

(2) That at unequal strengths the effect of interrupted light at 10 units is about equal to that of steady light at 2·5 units, and of steady light at 10 units to that of interrupted light at 40 units, the open sectors being $\frac{1}{4}$ of the disc.

	Interr. 10.		Steady 2·5.
Compare deflections	2, 4, 6, 8, 10,	with	13, 14, 15
	Steady 10.		Interr. 40.
,, ,,	1, 3, 5, 7, 9	with	12

particular experiment of which fig. 2 gives the results, the light was blocked off for seven-eighths of a minute and let on for one-eighth of a minute, during each successive minute, by means of an electro-magnet in the circuit of a battery and a mercury pool at which contact was made by a metal point fixed to a clock wheel and revolving once a minute. These instrumental details need not be minutely described; the apparatus may be inspected in the dark room; its plan is diagrammatically presented in fig. 13.

A simple inspection of fig. 2 shows us that the heights of the ordinates (*i.e.*, the magnitudes of effects) corresponding to an arithmetical increase of causative stimuli along the abscissa, do not form a straight line, but a curve, concave to the abscissa—a curve increasing by diminishing increments. It is similar in character to the classical Weber-Fechner curve given above.

I have also graduated the light by means of a revolving disc, with open sectors, the size of which could be varied. Fig. 3 exhibits the results of such an experiment.

This of itself is, I think, justification enough for an answer to the question formulated a few minutes ago—which I formulated in other terms two years ago—"Is the material sensificatory brain-change proportional to the internal stimulus, and the sensation some geometrical function of that change, or is the material change itself a geometrical function of the stimulus, and attended with sensation in direct proportion to its own magnitude?"[1] I then felt entitled to no opinion of my own, but quoted authorities *pro* and authorities *con* without adding note or comment. But looking to this result, considering that in fig. 2 we have external stimulus along the abscissa, internal stimulus along the ordinate, our judgment must be strongly inclined towards the second alternative.

II.—Let me at once turn your attention to a second item in the case, viz., to MUSCLE.

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[1] Introduction to "Human Physiology," 2nd edition, p. 550.

Considering muscle, not for its own special sake, but in a most general light as excitable living matter, of which the chemico-physical response to external stimuli is measurable, we may study as an instance of the relation between causative stimulus and excited effect, the relation in muscle between magnitude of stimulation and magnitude of contraction.

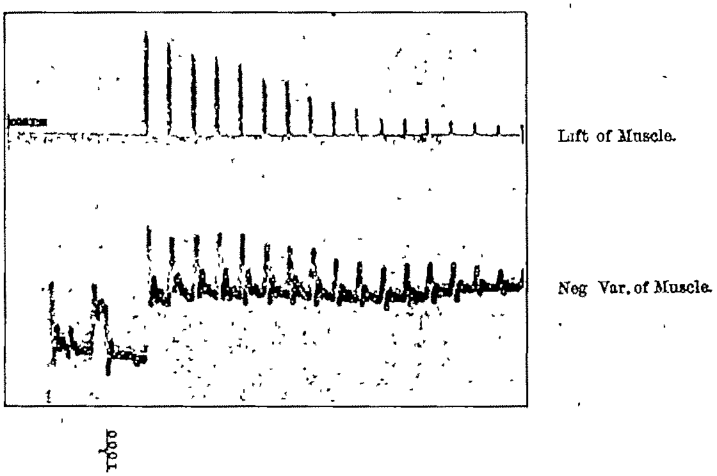

FIG. 4.

Simultaneous tracing of muscular contraction, and of the accompanying current of action. Galvanometer shunt $\frac{1}{80}$. Preliminary upward deflection by $\frac{1}{1000}$ volt.

We might do this galvanometrically, but the galvanometric sign of action in the case of muscle is simply a *replica*[1] of its mechanical response, and the latter is far more simply examined. I have urged elsewhere that as regards mechanical response, it is better to adopt an approximately isometric than an approximately isotonic method.[2]

---

[1] This is not, strictly speaking, correct; the electrical signs of muscular action are a far more delicate, and in some respects more accurate, quantitative indication than are the mechanical signs; moreover, the electrical response outlasts the *visible* mechanical response.

[2] "The Sense of Effort," BRAIN, 1891, p. 186.

An inspection of the two figures 5 and 6 shows that the general law, diminishing increments of effect (contraction), produced by equal increments of cause (stimulation), is similar to that already observed in the case of sensation, and in that of sensificatory change.

I do not now wish to dwell upon details, nor technical matters, nor historical matters, but before passing on to our next case I should like just to mention that the position here taken up has been considered and studied by other observers. Fick, Hermann, Preyer, Santesson, Cybulski and Zanietowski have handled the question, and Preyer in particular has considered it from the general point of view here taken up as indicated by the title of his monograph, "Das myophysisches Gesetz," Jena, 1894, the chief conclusion of which is to the effect that the shortening of muscle varies as the logarithm of the electrical excitation.[1] The other point

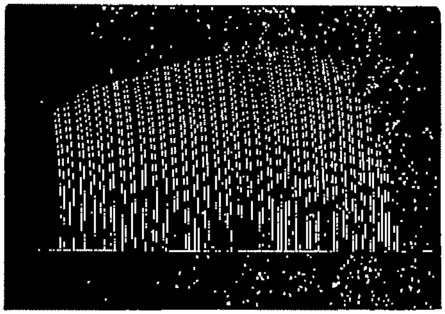

FIG. 5.
(To be read from right to left.)

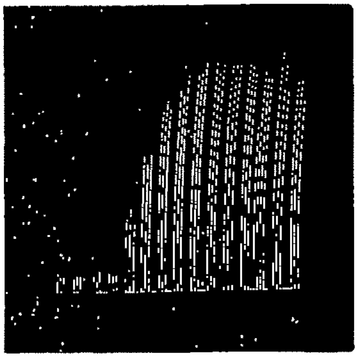

FIG. 6.
(To be read from left to right)

Contractions of a frog's gastrocnemius in fig. 6, caused by induction shocks of arithmetically increasing strength in fig. 5, caused by the discharges of arithmetically increasing quantity from a condenser.

Fig. 5 exhibits a curve of increasing effect with diminishing increments, viz., at once concave to the abscissa.

Fig. 6 exhibits an S-shaped curve of increasing effects, viz., at first convex then concave towards the abscissa.

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[1] And Dubois, in his admirable monograph on *Pholas Dactylus*, urges the same idea on still more cogent data, involving as they do both motion and sensation. In his account the magnitude of the "mouvement avertisseur" giving rise to the dermic phosphene, is a logarithmic function of the magnitude of stimulation, and the magnitude of the reflex retraction of the syphon symptomatic of sensation is proportional to the magnitude of the "mouvement avertisseur," which is the antecedent link in the sensificatory process.—"La Pholade Dactyle," Paris, 1892.

to which I wish to call your attention in passing is one that from a totally different standpoint has been insisted on by Prof. Weldon, and is illustrated in fig. 6.

The ordinates do not give from the outset a diminishing increment leading to a curve concave to the abscissa, but at the very outset increasing increments, and later only the usual diminishing increments. The curve formed by joining the summits of the series of contraction ordinates is S-shaped, beginning convex to the abscissa, continuing concave to the abscissa. And, as we shall see in a moment, a similar mode of commencement is apparent in the case of nerve (*e.g.*, in fig. 7).

III.—The third case to be considered is that of NERVE. Here (if I am not grossly deceiving myself, and have not fallen into some unsuspected form of instrumental fallacy) the results are of the highest interest. On nerve the curve expressing the relation between cause and effect is a straight line, the effect is proportional to the cause, at least within moderate ranges of excitation, exceeding, however, the upper limit of maximum functional effect, as gauged by muscular contraction. I hasten to add a short description of the conditions of experiment. The usual nerve-muscle preparation is laid across two pairs of electrodes; the exciting electrodes from an induction-coil are against the nerve midway between the leading-off electrodes to the galvanometer and the muscle, which in this case is preserved as an accessory indicator of function in the nerve. The electrical effects on the galvanometer and the mechanical effects on the muscle are simultaneously recorded on the photographic plate in a way that would be tedious to describe. It will be obvious if you will glance at the apparatus; its plan is diagrammatically presented in fig. 14. All precautions known to me for the avoidance of the two chief fallacies in such an experiment, viz., electrotonic currents and unipolar action, have been observed.

Fig. 7 exhibits the point I have just stated; the electrical effects on nerve consequent upon stimuli of magnitudes 1, 2, 3, 4, are in the proportion, 1, 2, 3, 4, a point that is still

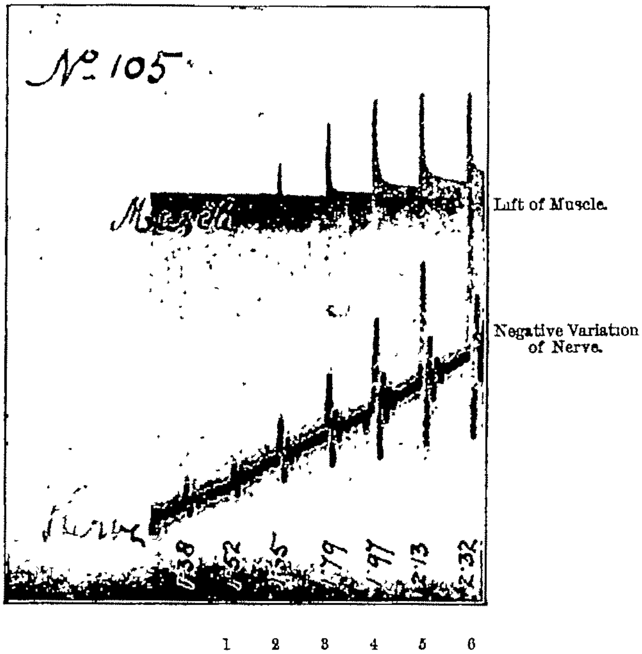

FIG. 7.

	Strength of Stim.	Nerve Neg Var.	Muscle Lift.
No. 1	1·52 units ..	2·5 mm. ..	0
„ 2	1·65 „ ..	5 „ ..	4
„ 3	1·79 „ ..	7·5 „ ..	9
„ 4	1·97 „ ..	12 „ ..	12
„ 5	2·13 „ ..	15 „ ..	12·5
„ 6	2·32 „ ..	19 „ ..	12·5

Simultaneous tracing of the galvanometric and muscular effects of nerve excitation. The nerve of a nerve-muscle preparation led off to the galvanometer by unpolar electrodes to its central end, is excited by tetanising currents at intervals of 1 min. for periods of $\frac{1}{8}$ min. No galvanometer shunt; each centimetre of swing = about $\frac{1}{10000}$ volt.

N.B.—The current of injury of the nerve was such as to deflect the spot downwards as regards this tracing; the upward inclination of the galvanogram indicates the usual decline of that current; the upward black lines across the galvanogram are due to the negative variations, the subsequent downward black lines are due to after-oscillations of the magnet and mirror. The plan of experiment is shown by fig. 14, but as printed the tracing is reversed, *i.e.*, the muscle shadow was south of the nerve deflection, and the current of injury was north (cut end of nerve to north terminal) and the negative variation south.

more evident in the diagram fig. 8, constructed from corrected data in continuation of the experiment, part of which is given in fig. 7.

The chief positive results have been laid before you, and will have to be matured by further experimental criticism. The main question of the Weber-Fechner relation between stimulation and sensation is touched by them at more than one of its facets. The more general question of the relation between physical cause and physiological effect has *ipso facto* been involved. And as in any consideration of phenomena *de tactu*, side-views of phenomena accessory to the main inquiry have presented themselves, and glimpses of imperfectly viewed relations—what may be termed sub-positive considerations—have also offered themselves to mind. In this connection the following points seem to me to be the more considerable:—

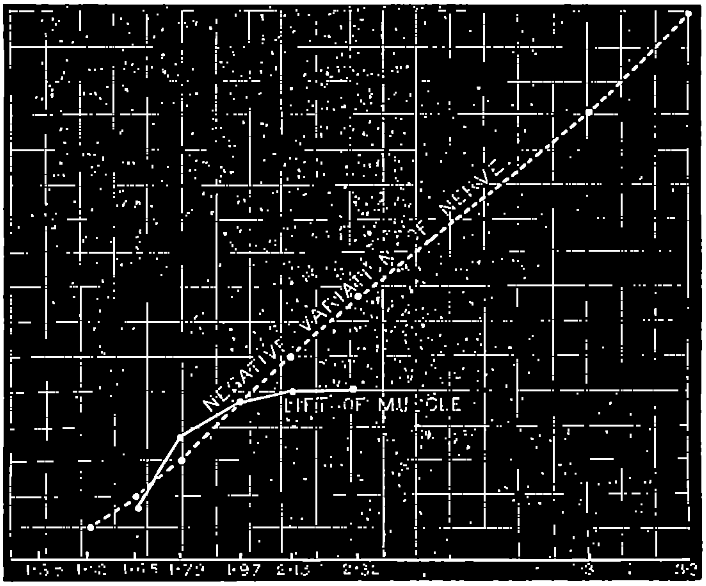

FIG. 8.

Stimulation units along the abscissa; electrical effects in nerve, and simultaneously observed mechanical effects of its muscle, along the ordinates.

(1) The S-shaped curve of muscle exhibited, *e.g.*, in fig. 6—is it peculiar to muscle, or to muscle as representative of living matter? Will it find itself repeated in a Weber-Fechner sensation curve? I am unable to say. I have no *data* of my own with regard to that most difficult investigation, viz., that of the sensation curve low down, as it is rising from the threshold of stimulation. But I would ask you to consider probabilities. Which should we prefer—to have maximum increments of sensation to equal increments of stimulation low down in the scale close to the threshold, or higher up in the scale within the working range of the stimulation experienced in ordinary life? The first alternative would, it seems to me, be characteristic of an almost intolerable state of hyperæsthesia in relation to the tumult of minute stimuli by which we are surrounded. I should prefer to be endowed with an S-shaped curve. And this is not merely a personal preference; there are one or two significant hints from various sides pointing towards the conclusion that an S-shaped curve is typical of the response of living matter, whether by quantity of sensation, or by quantity of motion, or by quantity of loss of motion, in response to external stimulation, to physiological accidents and to pathological injury.

It is only in the medium range of the scale of sensation that the logarithmic curve comes out. Hering and his pupils, working at lower ranges, found that the curve was approximately a straight line. Considering the enormous difficulties attendant upon such experiments, it is easy to believe that the initial tail of an S should present itself to the observer as a straight line. In the case of a muscle—if it is allowable to regard muscle as a representative of living matter, and the physico-chemical changes taking place in it in consequence of stimulation as analogous with the sensificatory physico-chemical changes taking place in the living matter of sensory organs—we have this S-shaped curve outward and visible. And on this same living matter—muscle—the decline at death and the decline in fatigue, as far as I have been able to judge, run this S-shaped course, only the **S** of the waning is as the **S** of the waxing reversed—as if viewed in a mirror.[1]

(2) The second most thought-arousing point is with regard to the electrical signs of nerve activity—of the nerve-fibre, not of the nerve-cell. I pointed out some ten years ago, quite independently of the observations of Wedenski and Bowditch, that nerve-fibre is practically inexhaustible, a force-transmitting, not a force-transforming organ, a passive physical conductor. I then made certain reservations as regards the experimental fallacy of electrotonic currents, which have been removed by further investigation, and the conclusion is at present an admitted one. Well, the conclusion being admitted, I do not think that it need surprise us to find in such an organ the physico-chemical effect simply proportional to the physico-chemical cause.

I hasten to add that it is only within a certain range—a range far exceeding, however, the physiological working-range of nerve—that the simple proportion holds good. Many more experiments directed to this particular point will be required before I shall be able to lay an entire curve before you with any justifiable degree of confidence. All I am entitled to say at present is that within what may be termed physiological limits, the curve expressing the ratio between cause and effect is a straight line; the sub-minimal and supra-maximal ends of that curve require further experimental determination; as far as my experiments have yet gone it has appeared to me that the beginning of the curve is convex to the abscissa, the end of the curve concave to the abscissa.

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[1] Without unduly insisting upon its possible significance, the fact is perhaps worth noting that an ordinary simple contraction of muscle due to a single stimulus, consists in each of its two parts of an S-shaped curve; in the period of ascending energy, the rise is by increments increasing, then diminishing, in the period of descending energy the fall is by decrements at first increasing then diminishing, giving ∫∿. The rising and falling effects have respectively their immediate cause in the muscle; it may be that each such cause is of uniform speed, in which case the effect is slow—quick—slow with uniform cause; or it may be that each such cause is itself slow—quick—slow, with its effect correspondingly slow—quick—slow; but even in this case there is an S-shaped relation between cause and effect, inasmuch as the immediate cause is itself an effect of a previous cause, the instantaneous stimulus provocative of decomposition of matter and evolution of energy, during the rise followed by what may be regarded as a negative state, provocative of recomposition and involution of energy during the fall.

Considering only that part of the nerve curve corresponding with the functional range between minimal and maximal muscular effects, we have a straight line, below which a short initial tail is convex to the abscissa, and above which, after a further prolongation of the straight line, the curve tends to become concave to the abscissa. An entire curve may then be considered as composed of three portions: (1) a short subminimal portion, convex to the abscissa, increasing by increasing increments; (2) a long straight middle portion, inclusive of the functional range between minimal and maximal muscular effect, increasing by equal increments; (3) an ultra-maximal portion far above the values of maximal functional effects, concave to the abscissa, increasing by diminishing increments.

It is not possible to exhibit these several features in any one line drawn to scale. To bring out the first portion of the curve the scale must be chosen very large, when of course the second and third portions are lost to view; to bring out the third portion the scale must be chosen very small, when of course the first portion is altogether indistinguishable. On an intermediate scale, the functional portion of the second portion is best shown, *e.g.*, in fig. 8. The first portion is visible in fig. 7. The third portion can be constructed from the numbers of fig. 9.

(3) A third point is subsidiary to the above. It will be most clearly exposed to you by the examination of an actual experiment (*e.g.*, fig. 9), viz., that far beyond that point where stimulation of the nerve gives a maximal effect upon the muscle, the effect upon the galvanometer of the electrical change in the nerve continues to increase. Due care was observed to exclude the unipolar and the electrotonic fallacies; the electrical effects were unchanged in direction with reversed direction of the excitation, viz., they were true "negative variations," the electrical concomitants of excitatory changes in the nerve. This being so, we are bound to recognise that the magnitude of an excitatory effect in nerve-conductors provoked by electrical excitation, is no index to the magnitude of functional disturbance within what may be regarded as physiological limits.

Method.—The most essential feature of the method employed in the above observations was the use of photo-galvanographic records, by means of an apparatus (galvanograph) already described some years ago in another place,[1] modified in the present instance by the substitution of a sensitive plate for the sensitive paper previously used. The

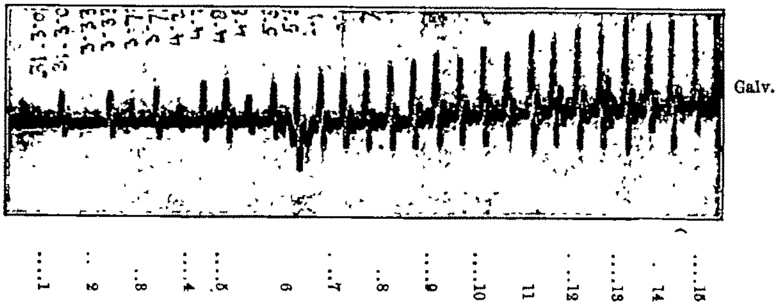

FIG. 9.

Continuation of Obs., 105. Galvanometer shunted $\frac{1}{10}$.

	Stimulation strength.	Galvanometric deflection.
No. 1	3·02 units	35 mm.
,, 2	3·33 ,,	40 ,,
,, 3	3·72 ,,	
,, 4	4·25 ,,	50 ,,
,, 5	4·80 ,,	
,, 6	5·50 ,,	60 ,,
,, 7	6·10 ,,	
,, 8	7·00 ,,	70 ,,
,, 9	8·20 ,,	
,, 10	9·60 ,,	85 ,,
,, 11	19·20 ,,	
,, 12	28·75 ,,	105 ,,
,, 13	38·30 ,,	110 ,,
,, 14	47·90 ,,	110 ,,
,, 15	57·50 ,,	110 ,,

N.B.—The values given for these galvanometric deflections are of necessity inexact, inasmuch as measurements of ordinates, the magnitudes of which have been tenthed by the shunt, and their subsequent multiplication by ten must give rise to a considerable error. But this error does not disguise the main fact apparent on simple inspection of the fig., viz., that the negative variation of nerve continues to increase with increasing strength of stimulation long beyond that strength which has produced a maximal muscular effect as shown by the preceding figure. The deflections to which numbers are not attached were caused by stimulation with reversed direction of current at the respective strengths indicated by the numbers, to exclude the suspicion of electrotonic currents.

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[1] *British Medical Journal*, July, 1885, p. 135.

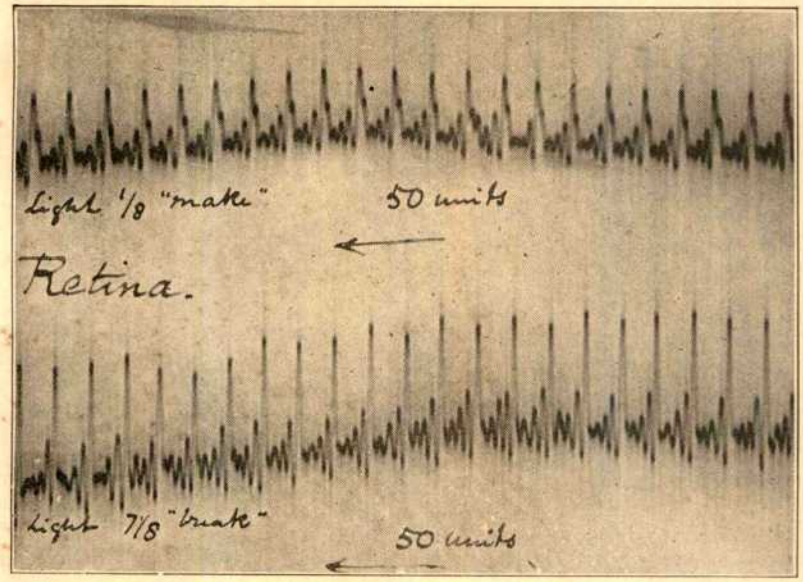


Fig. 10.

Currents of action from frog's retina six hours after excision of eyeball. The upper line is a series obtained by illumination for $\frac{1}{8}$ minute repeated each minute. Owing to the oscillation period of the magnet and mirror the deflection at "make" (about 4 mm.) is interrupted and augmented by the subsequent deflection at "break," the point of "break" is signified by the black spot of the large rise; the oscillations after the large rise are due to the swings of the magnet as it comes to rest. No galvanometer shunt. Each centimetre swing = about $\frac{1}{10000}$ volt. The lower line is a series obtained by illumination for $\frac{7}{8}$ minute repeated each minute. Only the "break" effects are visible, the "make" effects are not separately distinguishable. Oscillations of the magnet as before.

These series illustrate the facts that (1) the effects at "break" of light are greater than those at "make" of light; (2) the effects at "make" and at "break" do not sensibly diminish with repetition.

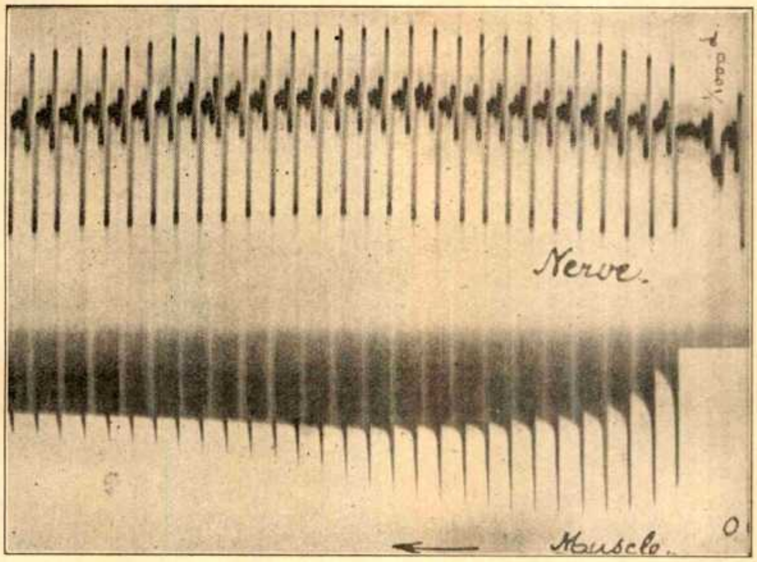


FIG. 11.

Simultaneous record of currents of action in nerve (upper line), and contraction of muscle (lower line), in accordance with the plan of experiment outlined in fig. 14. Tetanisation for $\frac{1}{2}$ min. repeated every minute. Galvanometer shunt $\frac{1}{5}$; preliminary downward or south deflection by $\frac{1}{1000}$ volt. Cut end of nerve to north terminal of galvanometer. Strength of coil 10 units. The electrical effects of nerve action do not sensibly diminish while the mechanical effects of the supplied muscle suffer the usual fatigue decline.

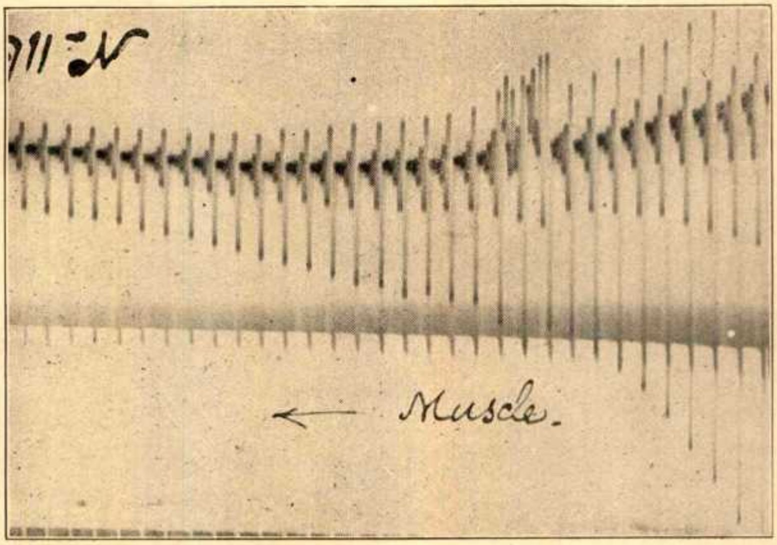
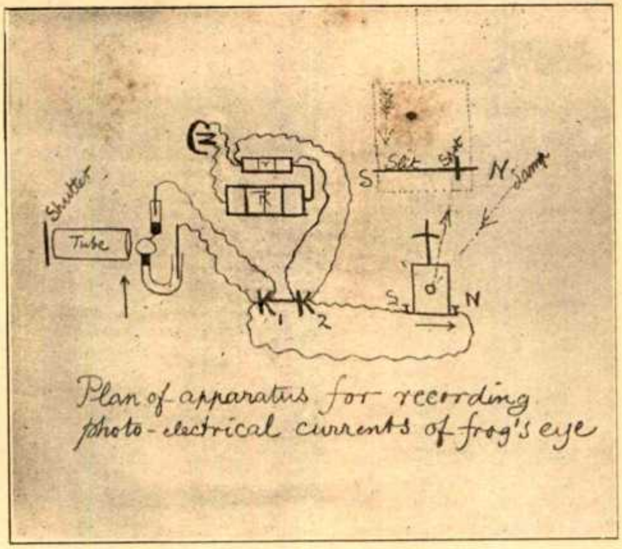


FIG. 12.

Simultaneous record of currents of action in muscle (large swings downwards, viz., south, the tendon being connected with the north terminal of the galvanometer, followed by the usual oscillations of the magnet as it comes to rest) and of the contractions of the same muscle. Galvanometer shunt $\frac{1}{9}$. Each centimeter swing = about $\frac{1}{1000}$ volt. Strength of coil 20 units. Tetanisation for $\frac{1}{8}$ min. repeated each minute.

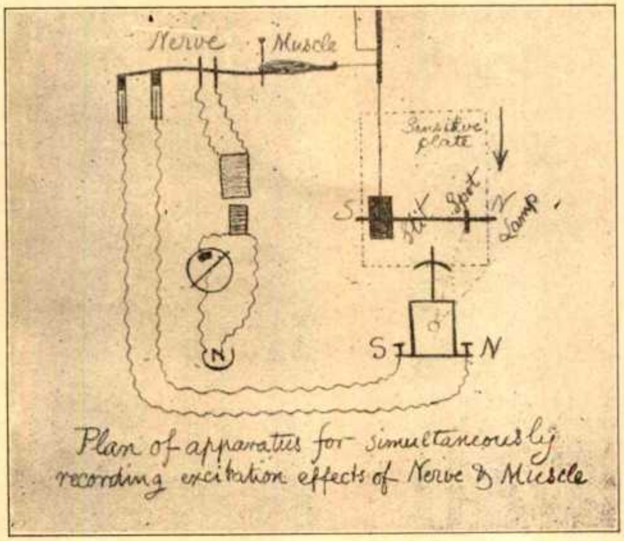
N.B.—The muscular contractions (downward black teeth from the black border) are at first overrun and rendered indistinct by the large deflections of the magnet. The irregularity in the series was accidentally caused by the fouling of a wire.

The electrical effects of muscular contraction decline with the decline of the mechanical effects. The former are a more delicate index of the state of muscle than the latter.



Plan of apparatus for recording photo-electrical currents of frog's eye

FIG. 13.



Plan of apparatus for simultaneously recording excitation effects of Nerve & Muscle

FIG 14.

entire complexion of enquiries of the kind is altered when the photographic method is adopted; points that escape the closest scrutiny in its absence, come into striking evidence, and doubtful readings are replaced by peremptory writings—autographs of the objects under observation.

Seeing that the intensity of light falling upon the eyeball varies inversely as the square of its distance, I prepared a scale graduated in units, in accordance with the following scale of numbers, choosing for the unit the $\frac{1}{100}$ part of a standard candle at a distance of 1 foot, or 1 candle at a distance of 10 feet.

DISTANCE.	STRENGTH.
1 foot	100 units.
1·15 ,,	75 ,,
1·41 ,,	50 ,,
2 ,,	25 ,,
2·29 ,,	20 ,,
2·58 ,,	15 ,,
3·16 ,,	10 ,,
3·33 ,,	9 ,,
3·53 ,,	8 ,,
3·78 ,,	7 ,,
4·08 ,,	6 ,,
4·47 ,,	5 ,,
5 ,,	4 ,,
5·77 ,,	3 ,,
7·07 ,,	2 ,,
10 ,,	1 ,,
11·5 ,,	0·75 ,,
14·1 ,,	0·50 ,,
20 ,,	0·25 ,,

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ON THE NATURE OF THE PHYSIOLOGICAL ELEMENT IN EMOTION.

BY PROF. A. E. WRIGHT, M.D.

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It is the signal merit of Prof. James to have recognised that the physiological element is the predominant element in the emotions, and that the problem of the nature of the emotions is essentially a physiological problem. The following study of the emotions consists in an attempt to apply to the solution of that problem the more adequate conceptions of the spinal centres and the somatic and visceral nerves which we owe to the researches of Gaskell.

Obviously the easiest way to approach the study of the emotions is to proceed by the method of physiological experiment.

An appropriate experiment would consist in taking a kitten and in noting the changes that occur in it when it is suddenly confronted with a strange dog. The emotional stimulus which would thus be applied would evoke a whole series of reflexes. The rhythm of the kitten's respiration would be altered, its heart-beat would be affected, the size of its pupils would be altered, and the hairs upon its tail would stand on end. All these would be reflexes upon visceral efferent nerves. Superadded to these involuntary visceral reflexes there would be reflex responses upon the semi-involuntary facial muscles; the lips would probably be drawn back so as to show the teeth. And, lastly, there would be reflex responses in the voluntary muscular system; the kitten would assume an attitude of self-defence.

The essential features of this emotional reaction would appear to be (*a*) the origination of the emotion in a violent sensory stimulus; (*b*) a resulting condition of extreme neural tension in the reflex centre; and (*c*) an outflow of neural energy, primarily, into the efferent paths associated with involuntary muscles, secondly, into the efferent paths associated with semi-involuntary muscles, and thirdly, into the efferent paths associated with voluntary muscle.

What holds true of the emotion which is evoked in the kitten by the encounter with the dog holds true of all other emotion. Wherever we have a condition of high neural tension, followed by reflexes upon involuntary muscle, we are dealing with emotion.

The descriptions which the poets give of emotion are in evident agreement with our definition. Two will suffice. We have Dante's "io ch'avea d'orror la testa cinta," and Juliet's "hood my unmanned [*i.e.*, untamed] blood bating [*i.e.*, beating as a hawk's wings beat] in my cheeks." Each of these descriptions of emotion tells of a condition of high neural tension, and of an outflow of neural currents into the involuntary muscular system. The "girdling of the head with horror" would indubitably be a function of the overflow of neural energy into the cervical sympathetic nerves, for Sherrington has demonstrated that the stimulation of the cervical sympathetic nerves[1] in the macaque produces the erection of a fringe of hair upon the head, and gives the monkey a terror-struck appearance. Similarly in the case of Juliet, the condition of emotion is associated with high neural tension and visceral reflexes. The latter result in tumultuous heart-beats and vaso-dilatation.

Moreover, the results of clinical observation accord well with our physiological inferences, for we find that heart disease, Bright's disease, jaundice, and dyspepsia are occasional sequelæ of profound emotional disturbance. The emotional reflexes have in these cases evidently been correlated with serious nutritional disturbances of the viscera.

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[1] Langley similarly showed that the erection of hairs on a cat's tail was brought about by the stimulation of the sympathetic fibres. On the other hand, as my friend Prof. Sherrington has pointed out to me, the labial vibrissæ in the cat are innervated, not by sympathetic fibres, but by branches of the fifth. This is exactly what we should expect *a priori*, for the erection of the vibrissæ of course occurs quite independently of any emotional reaction.

We must not, however, suppose that these visceral reflexes constitute the physiological essence of the emotion.[1] The physiological essence of the emotion is the high neural tension in the reflex centre, and not the visceral reflexes that result from that condition. For the psychological phenomena of any emotion precede the manifestation of that emotion, and they are coincident, not with the visceral reflexes, but with the antecedent neural tension in the reflex centres.

Having thus obtained a definition of emotion which conforms to the data of our physiological and psychological experience, we may pass on from this to consider the transformation of the reflexes that occurs when the stimuli which originally evoked emotions lose, as all stimuli gradually do, their emotional potency.

This transformation is a question of a replacement or the generalised somatico-visceral reflex by a specialised somatic reflex.

It is a transformation which is in gradual process all the time the child is growing up into the adult. In childhood the reflexes approximate to the primitive generalised type of reflex. There is as yet no differentiation into somatic and visceral reflexes, and every afferent phenomenon evokes a response both upon voluntary and upon involuntary muscle. In all such reflex responses there is therefore an element of emotion. When, however, the child grows older, the outflow of neural energy which occurs in response to the ordinary afferent impulses becomes more and more restricted to the somatic efferent channels. And gradually, hand in hand with this elaboration of the purposive voluntary action, the emotional visceral element[1] fades out from the reflex process.

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[1] We may perhaps note that the child does not discriminate between the essence of the emotion and its manifestation. The unsophisticated child will, for instance, say "Now you have made me *cry*." He will never say "Now you have made me *sorry*," for he does not attend to his psychological condition. He attends only to the results of his reflexes.

An example or two will serve to show what is involved in this substitution of a somatic for the primitive somatico-visceral reflex. When a tyro-sportsman goes out for the first time after game, the sight of a rabbit jumping up before him evokes a considerable volume of emotion. This expresses itself in a violent start, and in tumultuous heart-beats. When, however, the sportsman has become familiar with these sensations, and when he has practised the voluntary responses which are appropriate to such an occasion, the jumping up of a rabbit will no longer evoke an emotional reaction. It will evoke, instead, the purposive reflex responses which are associated with aiming and firing.

Similarly a surgical operation will evoke a considerable emotional reaction in any unaccustomed onlooker. But when a medical student sets himself to learn his surgical work, he learns to respond to such sensations with the appropriate somatic reflexes. And when the adaptation of these purposive voluntary responses to the sensory phenomena of the operating table has been achieved, the student, who now merits the title of "*surgeon*," finds that the nerve currents "no longer keep running down into his viscera."

We must, however, note that the education of the reflexes is never even tolerably complete. When any startlingly novel phenomenon—any sensation in short to which a voluntary reflex movement cannot be immediately adapted—presents itself, the neural tension will overflow with full force into the involuntary muscular system, and particularly into the respiratory system. We therefore speak of such a startling sensation as something "that takes the breath away."

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[1] The fading out of this emotional element from the reflex process is a frequent theme in Wordsworth's poetry.

It is, for instance, the theme of his "Intimations of Immortality from Recollections of Early Childhood." The "something that is gone" is the visceral reflex. The bringing down of all life to the lower level of the somatic reflex is the "closing of the prison house upon the growing boy." Childhood is "the hour of splendour in the grass and glory in the flower." Manhood is the time when the "celestial light" which originally illuminated every common thing has completely faded out "into the light of common day."

What has been effected, either by natural processes or by education is, therefore, not a suppression of the tendency of the neural tension to overflow into the visceral nervous system, but rather a diversion of the nerve outflow into the somatic efferent channels in the case of a certain number of constantly recurring sensations. And we must note that this more effective outflow through the somatic efferent channels goes hand in hand with a diminution of the neural tension in the reflex centres.

Very obvious physiological advantages accrue from this transformation of the reflex processes. *First,* the neural energy will no longer run to waste in visceral reflex movements. *Secondly,* when the visceral reflex is eliminated there will no longer be any danger of the visceral commotion, which is initiated by that reflex, reacting in an unfavourable manner upon the reflex centres. *Lastly,* when the high neural tension is abolished in the centre, the conditions will obviously be more favourable to the delicate adjustment of the reflex responses upon voluntary muscle. The advantages from the point of view of practical utility need not be further insisted on.

Considerations of utility do not, however, effect physiological changes. We have, therefore, still to seek for the effective physiological motive force which brings about this transformation of the reflexes. As a preparation for undertaking this task, we must, however, consider, *first,* the *a priori* necessity for some systematic control of the reflexes, and, *secondly,* the nature of the provision which actually exists in the organism for the exercise of that control.

The *a priori* necessity for some system of control becomes obvious when we consider that if each minimal stimulus were to evoke a separate reflex movement in an organism which was endowed with a sensitiveness at all approaching that of the human organism, life would be a mere chaos of muscular movement. If there were no storing away of nerve-energy there would be no continuity of purpose, and no possibility of making the present subserve the requirements of the future.

It is, however, precisely this continuity of purpose, this making provision for the future, this "looking before and after," which is the specially characteristic note of human life. And of all this an effective control of the reflex processes is the precondition.

In the case of the reflexes upon voluntary muscles, the control, which we have seen to be so essential, is exercised almost exclusively through the medium of the inhibitions. These are of course continuous in their operation. And so effective is this control that a stimulus cannot, even in the most sensitive organism, evoke a movement of voluntary muscle, unless it can first effect the lifting off a considerable dead weight of inhibition from the reflex centres.

In the case of the visceral reflexes inhibitory control plays a comparatively subordinate rôle. But on the other hand, the reflex centres are so constituted that no outflow of neural energy can take place into visceral efferent nerves until a very considerable neural tension has been achieved in the reflex centre. This provision operates as a very effective check upon the visceral reflexes.

The voluntary and the visceral reflexes are thus sharply contrasted. The voluntary reflex movements (*a*) correspond to low tension discharges from the centre (*b*) the mechanism which controls them is extraneous to the spinal reflex centre, and (*c*) the control of that mechanism can be relaxed. The visceral reflexes, on the other hand, correspond to high tension discharges from the reflex centre, and the mechanism that controls them is to a large extent intrinsic in the centre.

We have confirmation of this in the fact that a very different intensity of stimulus is required to evoke, on the one hand, a reflex on voluntary muscle, and on the other hand, a visceral reflex. We react with a movement of voluntary muscle to even the lightest touch, but when we react with a visceral reflex, it is always either a question of a very intense form of stimulus, or of a long series of stimuli which have gradually summated themselves in the centre. We may, for instance, contrast the facile culmination of our ordinary uninhibited somatic reflexes with the prolonged molimen which precedes the culmination of the reflex process in either the vomiting or the ejaculatory centre. It is always easy to initiate any voluntary muscular movement that may occur to us, while it is always difficult to call up an emotion *ex æquo*.

Again, we have clear indications of the high neural tension which is associated with the more important visceral reflexes in the fact that the summation of stimuli which leads up to the reflex action is attended with an outflow of energy along vasomotor and other visceral nerve channels. This is no doubt to be interpreted as a leaking[1] out of the nerve energy which is being accumulated at high tension in the reflex centre. We may take as an example the flushes and shivers and salivation which manifest themselves when the neural tension is rising in the vomiting centre.[2]

Lastly, the enormously greater neural tension, which is associated with visceral as contrasted with somatic reflexes, can be inferred from the intensity of the physiological urgency which precedes the culmination of a visceral reflex.

We here touch the central point of our problem. All high neural tension is attended with a sense of distress.[3] It is this physiological urgency, and not any calculation of utility which presides over the transformation of the condition of high neural tension which is associated with emotion to the condition of low neural tension which is associated with purposive action. It is, for instance, not a consideration of utility which urges on a nervous woman to try to learn to cross over a crowded thoroughfare without an emotional reaction. But it is the feeling of distress which is associated with the condition of high neural tension which she seeks to evade. It is with a view to compassing this object that she strives to respond with unhesitating purposive voluntary actions to the multiform stimuli of the street crossing.

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[1] This leakiness of the reflex centres must obviously operate as an effective drag upon the visceral reflexes. For wherever a certain interval of time is allowed to elapse between stimulus and stimulus the neural tension of the centre sinks to zero again, and the whole process of summation has to be taken under practically *de novo*. This is a fact which is in the experience of nearly everyone, not only after a rough cross channel passage, but also in connection with emotional disturbances in other centres.

[2] The distinguishing mark of what are spoken of in physiological text-books as "centres" (*e.g.*, the centre for vomiting, the centre for micturition, the centre for respiration) is the fact that these are high tension (visceral), as distinguished from low tension (somatic), reflex centres.

[3] It is this sense of general neural distress, and not actual pain, that is the true physiological opposite of pleasure. All neural distress is associated with a condition of high neural tension, and all such neural distress urges forward to the euphoria, which is associated with the condition of neural equilibrium. It is the rebound from the condition of neural distress to the condition of neural equilibrium which constitutes, as indeed Schopenhauer discerned, the essence of pleasure.

We have thus been able to discern what the compelling force is that brings about a gradual supersession of the generalized somatico-visceral reflexes of inexperience and childhood by the specialized purposive reflexes of experience and adult life. We have seen that it is not so much a question of substituting one variety of reflex for another, as it is a question of substituting a condition of low neural tension for a condition of high neural tension.

Now we cannot conceive any other means by which this transformation could be effected except by a more prompt and more adequate outflow of energy through the low tension somatic efferent channels. And such freer outflow of neural energy is almost certainly obtained by facilitating the somatic reflex responses of the spinal centre by a prompt relaxation of the inhibitions which are continuously passing down from the higher to the subordinate centres.

All these points in the physiology of the emotions can readily be illustrated if we allow ourselves to compare the complex of reflex centres, which Gaskell has taught us to group together under the concept of a "complete spinal segment," to a water cistern.

In accordance with Gaskell's scheme, such a water cistern would evidently be supplied by two inflow pipes, corresponding to the somatic and visceral inflow, and with two outflow pipes, corresponding to the somatic and visceral outflow.

Now these outlet pipes will be situated at different levels. The low level outlet will correspond to the efferent paths to voluntary muscle, and the high level outlet will correspond to the efferent paths to the visceral system. We have further to imagine, first, that the low level outlet was primitively partially obstructed by a valve, and secondly, that this valve has subsequently been geared up to a mechanism which can raise and depress it in such a manner as to completely free or to completely obstruct the flow of water through the lower outlet.

The primitive somatic and visceral reflexes of the child will now correspond to (*a*) a rapid inflow of water; (*b*) the development of a considerable head of pressure in the cistern; and (*c*) an outflow through the low level outlet, and, because of the insufficiency of this outlet, also (*d*) an outflow through the high level outlet.

The transformation of the reflex processes in the direction of a freer outflow into the voluntary muscular system will correspond to such more ample outflow through the low level outlet as will result from a removal of the inhibitory obstruction.

If we now make the further assumption that a considerable strain would be put upon the cistern by the pressure of the full volume of water, we shall be able to realise the utility of a provision for increasing the lower level outflow.

Our illustration will also bring it home to us that the undeniable utility of a provision for an ampler low level outflow is something totally distinct from a physical force such as would necessarily effect an alteration in the system of outflow.

Not only will our illustration make this plain, but it will supply us with something which is completely analogous to the physiological force which is ever tending to bring about a substitution of a somatic for a visceral outflow. The analogous force in the case of our cistern is obviously the pressure of the water, and we can easily conceive methods by which this pressure of water could be made to operate as a constant force in the direction of the removal of the valvular obstruction from the lower outlet.

Again, we can represent to ourselves the emotional results of violent, or sudden, or unfamiliar stimuli if we assume that the mechanism that controls the outflow through the lower outlet has only been adapted to the circumstances of the ordinary inflow, and that it is inadequate to control the rise of water in the cistern under the circumstances of any unusual inflow. Any such defect of adaptation would obviously bring about an outflow of water through the high level outlet.

Lastly, if we suppose our cistern to leak, and to leak through wider openings as the water pressure increases, we shall evidently constitute a further analogy between our water cistern and the spinal nerve centre.

ON THE FLOCCULUS.

BY ALEXANDER BRUCE, M.A., M.D.

THE flocculus becomes differentiated as a lobule, distinct from the rest of the cerebellum, at a very early period of intra-uterine life—almost contemporaneously, according to Michalcovicz, with the appearance of the middle lobe. Its peduncle also becomes comparatively soon myelinated, some fibres being so at the sixth month, and many more during the seventh month. This development occurs at the time when there are no myelinated fibres in the cerebellum, with the exception of the various constituents of the restiform body and the superior cerebellar peduncle. The peduncle of the flocculus, both at this early stage and throughout the rest of intra-uterine life presents such a striking contrast to the various strands in its neighbourhood that it is somewhat remarkable that so little attention has been paid to its connections as appears to have been done.

The terminations of this peduncle have been very variously described or altogether overlooked by recent writers on nervous anatomy.

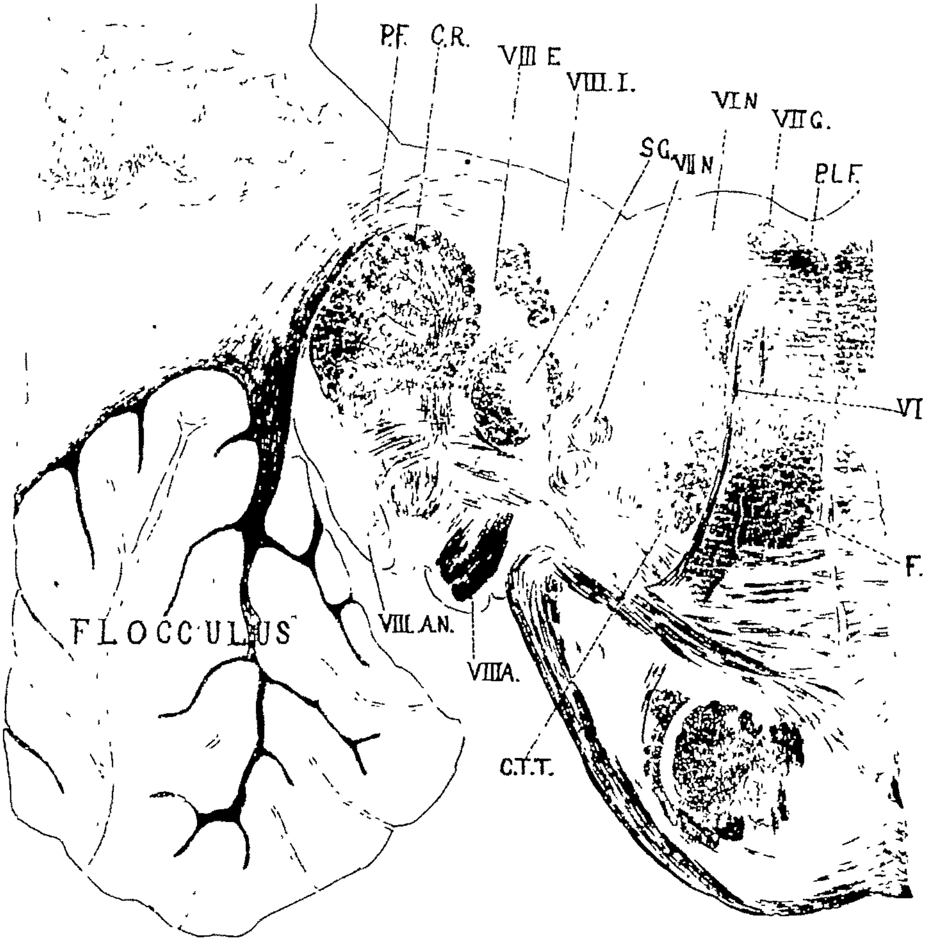
Thus Edinger makes no reference to it in his "Bau der central Nervös-organen," although Obersteiner ("Central Nervous System," English translation, p. 304) states that he (Edinger) traces it into the chief auditory nucleus of the opposite side.

Obersteiner (*loc. cit.*, p. 318) traces the flocculus into the brachium conjunctivum as far as the locus cœruleus. Vegas (*Archiv für Psychiatrie*, vol. xvi., p. 200) traces some of the fibres of the flocculus into the brachium conjunctivum. Bechterew, in "Die Leitungsbahnen im Gehirn und Rückenmark" (p. 137), traces its fibres along the roof of the fourth ventricle to the superior vermis. More recently Stscherbak (*Neurologische Centralblatt,* 1893, p. 227) traces part of its fibres to the vermis, along a path which passes between the inner division of the corpus restiforme and the nucleus dentatus; then along the outer side of the nucleus dentatus towards the nucleus globosus and embolus and the cortex of the vermis. Some of the fibres apparently form part of the commissure above the roof nucleus and pass to the opposite flocculus. The second portion passes inwards as far as the upper end of the chief nucleus, where it divides into fine fibres and disappears.

Stscherbak's paper is unfortunately not accompanied by any illustration, and his description is consequently somewhat difficult to follow, but it appears to me that the strand described as passing backwards to the vermis is probably not connected with the flocculus at all, but is really the bundle which passes from Deiter's nucleus along the lateral wall of the fourth ventricle towards the roof nuclei.

In 1892, in my "Illustrations of the Mid and Hind Brain," I stated that the flocculus passes inwards towards the lateral angle of the fourth ventricle and that the majority of its fibres pass inwards and enter into connection with the external and internal nuclei of the auditory nerve and also with the nucleus of the sixth nerve of the same side. The accessory nucleus is also connected with it by numerous fine fibres. It is probable that some fibres of the peduncle also enter into the nucleus dentatus and that another strand passes in an ascending direction along the side of the fourth ventricle. In fig. 7 the connection of the peduncle with the sixth nucleus is indicated. To have made this diagram complete, connections with both the auditory nuclei should have been shown. Indications of the position of the peduncle are shown in plates 9 and 10.

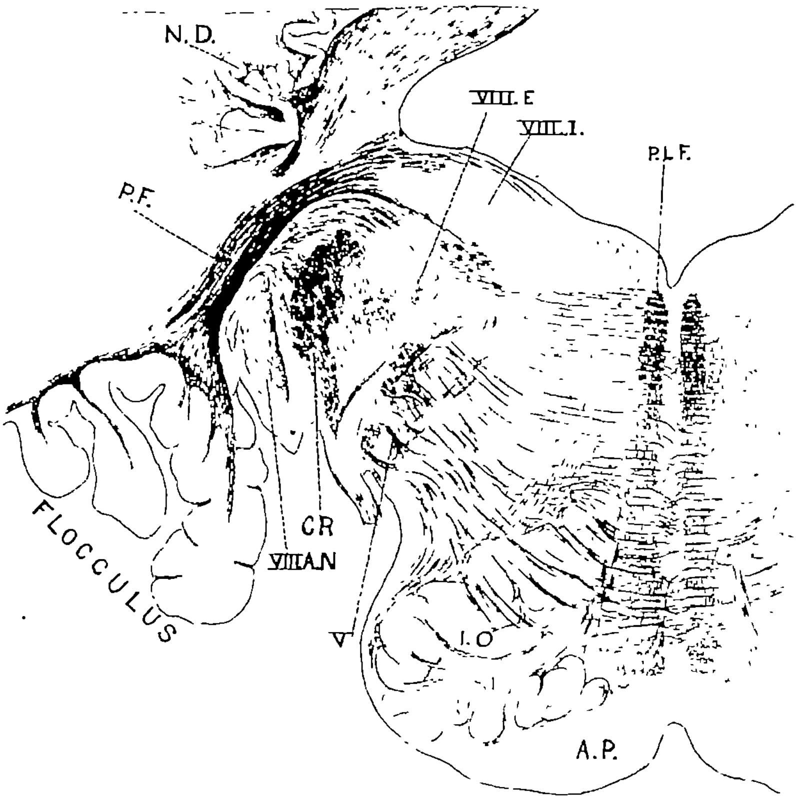
One reason for the variety of the opinions expressed is, I think, the manner in which the flocculus peduncle makes its entry into the side of the pons. If one makes a series of perfectly transverse antero-posterior sections of this region,



DESCRIPTION OF FIGURE 2.

Obliquely Transverse Antero-posterior Section of the Lower Part of the Pons made at an angle of 50 to 55° with the plane of the Raphé, the Mesial Part of the Section being at a higher level than the Lateral.

VII. N. = Nucleus of the facial nerve.
VII. G = Genu of the facial nerve.
VIII A. = Vestibular root of the auditory nerve.
VI. = Sixth nerve.
C. T. T. = Central tegmental tract.
F. = Fillet.
S. G. = Substantia gelatinosa, with the ascending root of the fifth to its outer side.
The other letters as in fig. 1.



DESCRIPTION OF FIGURE 1.

Perfectly Transverse Antero-posterior Section of the Upper Part of the Medulla and the Flocculus.

P.F. = Peduncle of flocculus passing towards the lateral angle of the fourth ventricle; the continuation of fibres underneath its floor and through the external auditory nucleus has been partly filled in from adjacent sections.

VIII. A. = Accessory nucleus of the cochlear root of the auditory nucleus.
VIII. E. = External auditory nucleus
VIII. I. = Internal ,, ,,
C. R. = Corpus restiforme.
V. = Ascending root of the fifth nerve.
N. D. = Nucleus dentatus. To its inner side part of the superior cerebellar peduncle.
P. L. F. = Posterior longitudinal fasciculus.
A. P. = Anterior pyramid.
I. O. = Inferior olive.

Note.—The level of the posterior part of the section is slightly higher than that of the anterior.

one sees the peduncle passing inwards and backwards along the outer aspect of the accessory nucleus of the auditory nerve (with which it has fine connections) and the external division of the restiform body towards the lateral angle of the fourth ventricle. On its reaching this angle it becomes forked and one part appears to pass inwards along the floor of the fourth ventricle, while the other part appears either to stop short, or to pass a little way upwards along the side of the ventricle, but owing to the angle which this part of the peduncle makes with the plane of perfectly transverse sections the fibres disappear from view more or less completely on reaching the margin of the ventricle. It is only by careful comparison of a series of sections that their further course can be traced (fig. 1).

With the view of further testing these conflicting views I have made a fresh series of sections in various directions.

Firstly, a series of perfectly transverse antero-posterior sections, at right angles to the plane of the raphé from a fœtus of 6½ months, showed that all the fibres of the flocculus that were myelinated passed inwards along the floor of the ventricle towards the auditory nuclei. None of these fibres apparently passed backwards towards the cerebellum.

Secondly, transverse sections made parallel to the plane of the floor of the fourth ventricle were made to ascertain the angle at which that part of the peduncle which passed inwards formed with the raphé. This angle was ascertained to be about 50° or 55°, the peduncle passing inwards and upwards as a strand of somewhat diverging fibres.

Next, transverse antero-posterior sections were made at an angle of 50° to 55°, with the plane of the raphé, and these confirmed in a very striking manner the results deduced from the comparison with a series of perfectly transverse sections. The peduncle (fig. 2) was seen to sweep round the accessory nucleus and the outer division of the restiform body, and to pass inwards almost, if not entirely, towards the auditory nuclei. Some of its fibres, to all appearance, end in the external nucleus. A larger number, however, pass under the floor of the fourth ventricle and are seen to break up within the internal auditory nucleus.

A few fibres can, I think, be traced into the nucleus of the sixth nerve, and some of them may possibly pass into the grey reticular formation of the same side. The myelination method does not appear to me to permit one to decide whether any of these fibres cross to the formatio reticularis or sixth nucleus of the opposite side.

In perfectly transverse sections made at a slightly higher level than fig. 1 some of the fibres of the flocculus appear to me to bend upwards towards the cells at the lateral angle of the medulla in a position which corresponds to that given by Bechterew as one of the nuclei of the vestibular roots of the auditory nerve. The fibres at this situation appear to me to break up into a fine network, from which again a comparatively small number pass backwards along the lateral wall of the fourth ventricle towards the vermiform lobe. Their manner of termination I have not been able to establish. It appears to me that the strand described by Stscherbak is not connected with the flocculus at all, but passes from the external nucleus of the auditory nerve to the roof nuclei of the cerebellum.

It would thus appear that the flocculus is an important central connection of, certainly, the nuclei of the vestibular root of the auditory nerve, the accessory nucleus of the cochlear root and, almost certainly, of the sixth nucleus. Its connections will no doubt be more fully traced by Marchi's method.

I should like to point out its closely related position to those somewhat rare tumours of the dura mater which arise from the neighbourhood of the internal auditory meatus, and also that the interference with the functions of this lobe caused by pressure upon it may be one of the causes of the vertigo which is associated with tumours in that situation.

THE
RESULTS OF EXPERIMENTAL DESTRUCTION OF THE TUBERCLE OF ROLANDO.

BY WILLIAM ALDREN TURNER, M.D.

(The experiments were made in the Neuro-pathological Laboratory, King's College, in association with Prof. Ferrier, F.R.S.)

In the course of an experimental investigation upon the conduction of sensory impressions in the spinal cord and brain, it was considered necessary to destroy, individually, the tubercles upon the posterior surface of the medulla oblongata. The symptoms following lesions of the clavate and cuneate tubercles have been elsewhere described;[1] in this communication the symptomatology of destruction of the *tubercle of Rolando* alone will be detailed.

It was observed that a lesion, thus localised, induced disturbances of sensation in the region of the fifth cranial nerve on one side, and upon the body and limbs on both sides.

It was found that experiments upon this tubercle caused a constant series of objective sensory phenomena; while those on the spinal cord showed that the effects of hemisection were uncertain and various, as indeed recent investigators have already pointed out.

The value of a localised lesion, such as that of the *tuberculum Rolando*, is obvious, in that being situated quite on the lateral aspect of the medulla, it may be destroyed without damage to the tracts passing up the opposite side; on the other hand, it is difficult to perform a hemisection of the cord without either directly or indirectly injuring structures situated across the middle line.

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[1] *Phil. Trans.*, vol. clxxxv., 1894, p. 755.

In the previous series of experiments upon hemisection of the spinal cord (BRAIN, 1891, p. 496), certain facts were noted, which showed that there did not exist entire uniformity in the symptoms following this operation; and others were at that time observed which were difficult to explain, but upon which subsequent, and more extensive, observation has thrown light. At the time referred to, the clip, as a means of testing cutaneous sensibility in animals, was not in general use; during the past few years, however, it has been almost universally adopted, and by some is probably relied upon exclusively, the earlier and more simple means being largely discarded.

Mott's[1] observations upon monkeys also show that considerable variety of symptoms followed this operation; sometimes the animal responded to cutaneous irritation more readily on the paralysed, sometimes on the non-paralysed side; and at others there appeared to be little difference between the two sides.

It would therefore seem from these, and subsequent experiments, that considerable variation exists in monkeys (and probably also in other animals) in the symptoms following hemisection of the spinal cord.

EXPERIMENTS.

Monkeys only were used in the investigation, and strict antiseptic precautions were invariably adopted. In every case chloroform was employed as an anæsthetic.

The tubercle of Rolando was exposed by an incision in the middle line of the neck, removal of the arch of the atlas and incision of the theca vertebralis. It is seen lying external, and somewhat caudal to, the *tuberculum cuneatum* and is easily recognised by its oval shape and greyer superficial appearance. It may be necessary, in order to bring the tubercle fully into view, to tilt the postero-inferior margin of the vermis cerebelli. The galvano-caustic knife was used to destroy the tubercle.

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[1] Mott, *Phil. Trans.*, 1892, vol. clxxxiii., B.p.

In the experiments to be presently recorded, the clip used had a more powerful grip than that now adopted in the laboratory. It cannot therefore be relied upon as a means of testing *slight* defects of tactile sensibility.

EXPERIMENT I.—Mac. rhœsus, April 23, 1892.

After recovery from the immediate effects of the operation, in which the *left* tubercle was destroyed, the monkey presented the following symptoms:—paresis of the left arm and leg, and insensibility to all forms of stimulation of the left cornea, and left side of the face and forehead; with temporary narrowing of the left palpebral fissure and a less prominent state of the left eye-ball.

On the day following the operation the animal was prostrate, and it was difficult to determine with certainty the state of cutaneous sensibility. On the third day, however, recovery had taken place, and the following symptoms were observed: there was no obvious evidence of the presence of tactile sensibility on the left limbs or left side of the body, for the animal paid no attention to gentle touching, ruffling the hair, or dropping water on this side, nor was the left hand or foot withdrawn when dipped into cold water, while a response was at once obtained when the right arm and leg were similarly irritated.

When the *clip* was attached to the left limbs, the monkey struggled and tried to bite, but made no attempt to remove it; on the other hand, when similarly attached to the right side there was no struggling, but the clip was at once pulled off.

Pricking gently the left side of the body, the left limbs and the right side of the face, invariably produced evidence of discomfort, for the monkey would struggle or bite, but no such response was obtained from the right lower limbs or the left side of the face; when the right arm was pricked the monkey occasionally gave some slight sign of sensation by biting or withdrawing the limb, but here it was more difficult to exclude possible vision.

Five days later there was obtained evidence of a sense of touch on the left or paretic limbs. The animal was on several occasions seen to scratch the place on the left arm where it was gently tickled.

The monkey remained in this condition without any very obvious change in the symptoms until June 22, when it was killed under chloroform. The cornea remained throughout anæsthetic and did not show any evidence of opacity or ulceration. There was only at the outset a temporary conjunctivitis and slight œdema of the eyelids.

Autopsy.—The lesion involved the tubercle of Rolando throughout its extent, the actual amount of destruction being shown in figs. 1 and 2.

In addition to the lesion of the tubercle, the adjacent structures were involved indirectly by extension of traumatic influences. The structures thus implicated were—the clavate and cuneate tubercles only in their proximal parts, the pyramidal tract as it bends from its decussation into the lateral column of the spinal cord, and the postero-lateral part of the grey reticular formation. The cicatrix, however, was chiefly confined to the gelatinous substance forming the tubercle of Rolando, which was completely destroyed.

EXPERIMENT II.—Mac. sinicus; April 26, 1892.

In this case also the *left* tubercle was destroyed. As a considerable quantity of blood was lost at the time of the operation, the monkey remained weak for four days. On regaining strength on the *fifth* day, the following were observed: a response to tactile impressions was obtained from both sides; either foot or hand was withdrawn from cold water, and an object was grasped when inserted into the palms or the soles. When the clip was applied to the right limbs, it was immediately withdrawn by the monkey, but there was little or no evidence that this was felt as a painful sensation, for there was no struggling or attempts at biting, which invariably occurred when the clip was attached to the left limbs. At times also the clip was localised on this side, more especially when applied to the buttocks and about the root of the tail.

Paresis of the left arm and leg, and anæsthesia of the left face, forehead and cornea existed.

This condition remained practically unchanged during the next fortnight. At this time the paresis had to a large extent passed off. Localisation also of tactile impressions was distinctly present on the left side, although the response was not so active as on the right.

The monkey was killed on June 9, 1892. By this time the paresis had disappeared, with the exception of loss of power of the finer movements of the left hand. The cornea was not now completely anæsthetic, and there was no evidence of any ulceration or opacity.

Autopsy.—The lesion in this case does not appear to have been so complete as in the previous experiment. Owing to some error in preparing the brain for microscopic examination, it has been found impossible to reproduce a photograph of the lesion; but from a study of the sections obtained, it was seen that the structures implicated were: the tubercle of Rolando, the top of the left crossed pyramidal tract, the adjacent cuneate tubercle and the dorsal part of the left grey reticular formation.

EXPERIMENT III.—"Sooty" monkey, male; October 29, 1892. Destruction of the *right* tubercle of Rolando.

After recovery from the operation the monkey presented considerable disturbance of equilibrium; there was a tendency to roll towards the right side, and it was unable to sit up without support. There was paresis of the right arm and leg; the right pupil was smaller than the left, and the right cornea and side of the face were anæsthetic. Gentle cutaneous irritation of the left limbs and left side of the body always caused the monkey to scratch the part so irritated, but no evidence of the presence of tactile sensibility was obtained on the right side. Pinching, pricking and the clip were invariably localised on the left side but not on the right.

The above was the only observation made upon this monkey. From the fact that it was suffering from diarrhœa at the time of the experiment, it did not long survive the operation.

Subsequent examination of the site of the lesion showed that the tubercle of Rolando was destroyed, and as in the previous cases the pyramidal tract, the adjacent portions of the grey recticular formation and *funiculus cuneatus* were slightly implicated.

EXPERIMENT IV.—Mac. rhœsus; November 22, 1892.

In this case the *right* tubercle was destroyed. On the day after the operation the monkey presented the following symptoms: paresis of the right arm and leg; there was no obvious evidence of the presence of tactile sensibility on the right limbs, as tested by gentle ruffling of the hair and the dropping of water; while similar irritation was at once localised on the left side by scratching. It bore with equanimity pricking on the left side, which caused active manifestations of feeling on the right. When the clip was applied to the right foot, struggling at once ensued; when attached to the left there was no struggling, but the animal at once withdrew it; it never attempted to withdraw the clip from the *right* foot. There was anæsthesia of the right cornea and right side of the face, and the right pupil was smaller than the left.

Two days later there was still no clear evidence that the monkey was aware of dropping water, or gentle rubbing of the skin of the right arm or leg. As evidence of analgesia of the left side may be mentioned the fact, that the monkey leant this side against the hot plate of the stove without apparent discomfort.

At the end of the week, however, there was no doubt that the monkey was aware of the gentlest touch on the right leg and right side of the body; it moved away from the source of irritation, but it did not localise by scratching, although it looked round to the place of irritation.

Six weeks after the operation the monkey was in good health. Although the right arm was still paretic, the leg had, to a great extent, recovered. It bore on the left limbs the pressure of forceps, which occasioned struggling and discomfort when applied to the right. The right cornea remained clear throughout, but later on its sensibility and that of the right side of the face was merely defective as compared with the opposite side. The monkey was killed on January 23, 1893, without any further change in the symptoms having occurred.

Autopsy.—The lesion, in this case, was practically the same as that in the first experiment. The amount of destruction has been reproduced in figures 3 and 4. At the lower part (distal) of the lesion, the only structure destroyed was the gelatinous substance of Rolando; while in the upper part, in addition to this, there were involved, and partly destroyed, the cuneate tubercle and the adjacent dorsal part of the grey reticular formation. But here again, the cicatrix produced by the cautery occupied the position of the *tuberculum Rolando.*

REMARKS.

The effects of localised destruction of the tubercle of Rolando, as observed in these experiments, may be divided into two sets; firstly, defects of sensibility over the distribution of the fifth cranial nerve on the side of the lesion; and secondly, alterations in the sensibility of the body and limbs on both sides.

(1) The phenomena over the distribution of the trigeminal nerve consisted in defect or abolition of all forms of sensation in the cornea, the skin and the mucous membranes supplied by that nerve. The loss of sensibility thus obtained, was due to the destruction of the so-called "ascending" trigeminal root, where it lies upon, and forms the superficial white stratum of, the Rolandic tubercle. It was specially observed in those cases, that, although the cornea remained anæsthetic throughout the life of the animal after the operation, in no case were "trophic" changes noted either in the form of corneal opacity, ulceration or ophthalmitis. This subject, however, has been submitted to more extensive experimental investigation, and brief reference has been already made to it.[1] The details of these experiments will be given in another communication.

Among other and minor symptoms noticed, were narrowing of the pupil on the side of the lesion, and in one experiment a very decided narrowing of the palpebral fissure on the same side. The existence of pupil-dilating fibres in the ophthalmic branch and sensory trunk of the trigeminus was thus confirmed, and their presence in the so-called "ascending" root has been established by these experiments.

(2) Coming now to the more important physiological question concerning the sensory phenomena in the body and limbs after this lesion, it will be noticed that the symptoms present a marked degree of uniformity.

The general statement may be made that, *on the side of the lesion*, there was temporarily after the operation no apparent evidence of the presence of a sense of touch, or of localisation, although in one case (Experiment II.), a sense of contact appeared to be present, but defective; while the sense of pain, as such, was in every instance retained. In the second experiment, however, the symptoms were not observed until the fifth day, by which time some recovery had probably taken place. *On the opposite side*, tactile impressions were invariably localised, while there was loss or defect of the sense of pain, as such, although the site of irritation, whether this was effected by pricking, or by the application of the clip, was accurately localised. On the side of lesion there was weakness of the voluntary movements of the arm and leg, which, on subsequent examination, was found to be due to injury to the fibres of the crossed pyramidal tract under the tubercle of Rolando (*vide* figures).

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[1] Ferrier, BRAIN, 1894, p. 1.

In order to compare these results with those of hemisection of the spinal cord by the same methods, the following two experiments were made.

EXPERIMENT V.—Section of the *left* side of the spinal cord. Mac. cynomolgus; June 11, 1892.

On recovery from the anæsthetic, the monkey was found to be paraplegic and to pay no attention to sensory impressions of any sort on either lower limb. There was no general shock, as it was otherwise active and took food well. This condition remained until the *third* day after the operation. When tested on this occasion, the monkey accurately localised the clip, pricking and gentle scratching of the skin of the *right* lower limb and right side of abdomen, but it gave no sign of pain or discomfort. It paid absolutely no attention to any form of irritation on the left lower limb. Both legs remained completely paralysed. The temperature of both soles was nearly equal: left sole 89·5° F., right sole, 90·5° F. The knee-jerks were equal and apparently normal. At the end of a week slight movements of flexion at the right hip-joint were detected, but the state of sensation remained as before. Although the animal invariably localised, where it was touched or pricked on the right lower limb, there was no evidence by struggling or otherwise that the latter was felt as painful. The left lower limb remained completely paralysed, analgesic and anæsthetic until the death of the animal six weeks after the operation.

Autopsy.—The lesion was made between the sixth and seventh dorsal nerves. Sections taken from the seat of lesion after hardening in Müller's fluid and staining by the Weigert-Pal method showed that the cicatrix extended across the middle plane. The whole of the left side of the spinal cord was divided and the incision filled with cicatricial tissue, which extended across so as to obliterate the posterior column of the opposite side, and, to some extent, the posterior horn of grey matter. The opposite lateral and anterior columns and the anterior horn were unaffected (fig. 5).

EXPERIMENT VI.—Section of the *left* side of the spinal cord in the upper dorsal region. Mac. sinicus; January 24, 1893.

On the day after the operation the monkey was submitted to careful testing, and the following facts were observed:—There was complete paralysis of the left leg; the repeated prick of a needle and the application of the clip were accurately localised upon the *right* leg, but no struggling or other evidence of the appreciation of painful sensation was observed, such as invariably happened when the *left* leg was pricked or the clip applied. On this side struggling followed pricking and the clip, but there was no attempt at localisation on the left lower limb. There was no clear evidence of a sense of contact on either leg, other than that already noted. Tactile impressions were readily detected on the arms.

One week after the operation, however, definite evidence was obtained of a sense of touch on the *right* foot, for when gently touched with the finger or when blown upon, the foot was withdrawn, and at times the monkey would scratch the place so irritated.

Three weeks after the operation the *left* lower limb remained completely paralysed, nor was there any evidence of the presence of a sense of touch upon this limb. Pricking and the clip gave active manifestations of feeling, but without localisation, while on the *right* side, although impressions both of touch and pain were localised, there were no signs that the latter was felt as such.

Autopsy.—The lesion itself was an almost exact hemisection, and is shown in fig. 6. With the exception of the mesial part of the anterior column, the whole of the left side of the cord was formed of the cicatricial tissue. The secondary degenerations also indicated an almost complete hemisection, for with the exception of a few scattered black spots (by Marchi's method) in the antero-lateral superficies of the right side, the resulting descending degeneration was limited to the lateral and antero-lateral columns on the side of the lesion.

Closer analysis of these two experiments shows certain interesting features. The symptoms immediately following the operation in Experiment V. were those of a complete transverse section of the spinal cord, both as regards motion, sensation, and the knee-jerks. On the third day recovery of tactile sensibility took place on the side opposite the lesion, but the state of motion remained defective for a longer time. To look carefully at the actual amount of destruction of the cord which had taken place, it will be seen that, in addition to the hemisection of the left side, the whole posterior column and the greater part of the posterior horn of grey matter on the right side, has been replaced by cicatricial tissue; while of the remainder of the grey matter, the anterior horn and adjacent tissue remains unaffected. So far, therefore, as the posterior region of the cord is concerned, the lesion is, to all intent, bi-lateral. This no doubt explains the bi-lateral anæsthesia and analgesia, which was temporarily observed. The early return of the sense of touch on the side partially destroyed, indicates that the tracts subserving touch may have a more general distribution throughout the grey matter than those of pain, in which case a greater amount of destruction is necessary to abolish tactile sensibility—a view which may explain the dissociated loss of painful sensibility in syringomyelia.

The symptoms observed in these two cases, especially Experiment VI., differed to some extent from those previously recorded after hemisection of the spinal cord (BRAIN, *op. cit.*), as well as from those observed by Mott[1] after a similar operation.

They emphasise what had been previously observed, and what was very apparent in Mott's experiments, viz., the removal of the clip only from the side opposite the lesion. It is difficult to state what form of sensation the application of the clip conveys, but it has been found from an extensive series of observations upon the production of sensory disturbances in monkeys, that the removal of the clip goes *pari passu* with the existence, but especially the

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[1] Mott, *Phil. Trans.*, *op. supra cit.*

localisation, of tactile sensibility, ascertained by other methods, whatever be the state of the motor functions. The removal of the clip from a paralysed leg was well illustrated in Experiment V., in which, as a result of hemisection, both legs were paralysed. The monkey, however, accurately localised the clip, on the side opposite the lesion.

It is necessary to point out, that although the clip was removed from the limbs on the side opposite the lesion, both after hemisection and after destruction of the tubercle of Rolando, there was no evidence of a sensation of pain as such. Neither by struggling nor otherwise did the animal give any evidence of this; while with the exception of the complex Experiment V., the application of the clip invariably gave rise to struggling on the side of the lesion. In both series of experiments the evidence bearing upon the loss of the sense of pain on the side opposite the lesion was most convincing. The sense of touch, however, appears to be differently conducted. In none of the experiments was there any clear evidence, at least temporarily, of the presence of a sense of touch on the side of lesion, while it was invariably shown to be present on the opposite side.

There are few experiments upon the region of the Rolandic tubercle, a record of which I have been able to obtain. Three are recorded by Herzen and Loewenthal.[1] Their experiments were performed on cats, but instead of destroying the tubercle of Rolando, they made an incision through the posterior column and the dorsal part of the lateral column on one side, at the junction of the spinal cord and medulla oblongata. The consequent disturbances of sensation do not seem to have been very minutely tested, but the general result is in harmony with those recorded in this paper. For example, in the first experiment, one month after the operation, the animal bore the contact of a piece of ice on the paws on the side of the lesion without any sign of sensation, but at once withdrew them when ice was applied to the opposite side.

In the second experiment sensation appeared to be less active on the side of the lesion than on the opposite side.

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[1] Herzen and Loewenthal, *Arch. de Physiol*, 1886, p. 260.

In the third case, a more energetic response followed pinching the toes of the paw on the side of the lesion than on the opposite side; while, as in the first case, one month after the operation, the limbs on the side of the lesion could be immersed in ice water without the slightest sign of sensation, while those on the opposite side were withdrawn on the first contact with the water. Further, no notice was taken of touching the hind limb on the side of lesion, while that on the opposite side was at once moved.

From this it appears that tactile sensibility, chiefly ascertained by the contact of ice, was abolished on the side of the lesion; painful sensation, on the other hand, tested by pinching, was defective on the side opposite the lesion.

Bechterew[1] also records an experiment which he performed upon a dog in this region. By passing a knife through the occipito-atloid foramen, he was able to divide the following structures on the right side — the greater part of Burdach's column, the fibres of the posterior decussation, the outer part of the reticular formation, as well as the gelatinous substance of Rolando and the ascending trigeminal root. As a result of this lesion he found, that the right side of the face, mouth, tongue, and the right cornea were anæsthetic and analgesic; and that the tactile sensibility on the right limbs was distinctly defective compared with the left, the sense of position of these limbs also was lost. On the left limbs the animal responded to the slightest cutaneous irritation, but made no response to painful impressions. The motor power of the limbs was not materially altered.

I have been unable to find the record of any case of disease or injury in man at this locality, which was followed by a *post-mortem* examination. There are two cases, however, in which the symptoms point to a lesion at or about the junction of the spinal cord and medulla oblongata.

(1) *Bechterew's Case.*[2]—The symptoms were observed one year after a bullet wound was inflicted on the left side at the level of the atlas; on the *right* side there was anæsthesia of the face, paralysis, eventually becoming merely weakness,

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[1] Bechterew, *Neurol. Centralb.*, 1894, p. 298.

[2] Bechterew, *Neurol. Centralb.*, 1893, p. 457.

of the right arm and leg, dulling of the sense of touch and complete palsy of the "muscle-sense." On the *left* side there was paresis of the arm and leg, which eventually disappeared, loss of the senses of pain and temperature, dulling of the sense of touch, but retention of the "muscle-sense."

In this case, as the motor symptoms show, the lesion was not completely unilateral, a fact which may account for the dulling of tactile sensibility on both sides. The condition of painful sensibility on the right side is not stated; nor is a definition given of the term "muscle-sense." If it may be interpreted as the sense of position, then its retention on the side on which tactile sensibility was least affected, and associated with loss of painful sensibility, is in close harmony with the recorded experiments upon the Rolandic tubercle.

(2) *Steiglitz's Case.*[1]—The *right* side of the face and right cornea were completely anæsthetic; the right side of the body, though paralysed, was sensible to the slightest touch, the pressure of the bedclothes being almost painful; but the sense of position of the limbs was uncertain. On the *left* side there was anæsthesia to touch, pain and temperature, but there was no ataxy of the left limbs.

In this case the association of anæsthesia of the face and cornea, with altered sensibility of the body and limbs, suggest a lesion above the second cervical nerve root, and in close proximity to the tubercle of Rolando. From the complete decussation of the senses of touch and pain, it is more probable, however, that the lesion is higher up in the medulla oblongata.

The original experiments recorded here, taken in association with those of Herzen, Loewenthal and Bechterew, show that at the medullary-spinal junction the paths conducting the sensations of touch and pain are represented on both sides; the path for painful sensibility being crossed, and that for touch both crossed and uncrossed.

It would appear, however, that lower down in the spinal cord there is no absolutely constant distribution of the fibres entering by the posterior nerve roots. Sometimes it was found experimentally that all forms of sensation were abolished on the side opposite the lesion, or again, that one form (touch) was abolished on the side of lesion, and another (pain) on the side opposite.

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[1] Steiglitz, *Neurol. Centralb.*, 1893, p. 145.

Coming, however, to the spinal-medullary junction, we find a more uniform method in the arrangement of the sensory tracts. At this level the path of painful impressions has entirely decussated, while those of touch are passing up more or less on both sides. In course of time after the operation, some recovery takes place, and we find the animal, whose Rolandic tubercle has been destroyed, again localising cutaneous impressions within a few weeks after the operation.

But if we now consider what are the probable paths of sensation, we find valuable evidence from the experiments on the Rolandic tubercle, taken in conjunction with Experiment V.

In the first series, the lesion was, for all practical purposes, limited to the gelatinous substance and posterior horn, and the symptoms were, loss of touch on the same side and loss of pain on the opposite.

In Experiment V. both posterior columns, one posterior horn completely and the opposite horn *partially*, were destroyed, and the symptoms were complete anæsthesia and analgesia on both limbs, but after the third day sensibility to touch returned on the side on which the posterior horn was only partially involved.

One may therefore conclude that the posterior horns contain the fibres, which subserve the cutaneous sensibility, rather than the posterior columns, which in the spinal experiment were both completely destroyed, and in the tubercle experiments only partially involved, and that by secondary traumatic rather than direct influence.

An examination of the figures shows that the gelatinous substance of Rolando, with the subjacent posterior horn, has been chiefly destroyed, although neighbouring structures have been, to a slight extent, secondarily involved. From the results of the experiments, it would appear that the gelatinous and spongy substances of the posterior horn may be of greater importance in the conduction of sensory impressions than has hitherto been suspected.

When examined histologically by the Golgi method, the gelatinous and spongy substances of the tubercle of Rolando are found to contain a large number of fine nerve fibres and their end-tufts, which the observations of Kolliker[1] and Held[2] have shown to be, in the medulla, the axis-cylinder processes and collaterals of the cells of the Gasserian ganglion. In the spinal cord a similar structure is observed, being in this case the terminal nucleus of certain of the posterior root fibres. Situated in the substantia spongiosa, in close relation to the end-tufts, are large multipolar nerve cells, whose axis-cylinder processes pass into the tegmentum medullæ.

It has also been shown that section of the sensory trunk of the fifth nerve proximal to the Gasserian ganglion, or of the so-called "ascending" trigeminal root in the medulla, was followed by degeneration and atrophy of the fibres, which pass through the gelatinous substance and of the network which these fibres form in the spongy substance of the posterior horn. No atrophy of the cells, on the other hand, was detected in this situation.[3]

What the further course of the sensory trigeminal fibres may be, is more difficult to state. Owing to the interruption of degeneration, which occurs in the substantia spongiosa, no direct observations by this method have been recorded; but the observations of Kölliker and Held point to a passage across the raphé in the medulla and pons of the axis-cylinder processes of the cells of the substantia spongiosa.

This crossed relation of the sensory trigeminal fibres, as well as of the sensory fibres from the body and limbs, was corroborated by an experiment, where, after hemisection of the tegmentum pontis, anæsthesia of the opposite half of the body was detected.

As elsewhere shown (*Phil. Trans., op. cit.*), destruction of the nuclei of Goll and Burdach was not followed by any appreciable defect of common sensibility in the body or limbs; and Mott (BRAIN, 1895, p. 11) has more recently stated, that it was impossible to make out anything definite regarding sensation after severence of the fibres leaving these nuclei. Now as these nuclei give origin to the fibres forming the fillet of the opposite side, it is impossible to support the view that the fillet is the tract of ordinary cutaneous sensation, at all events in the lower (distal) portions of the medulla oblongata. Moreover, there are cases on record of hæmorrhage into, or softening of, the tegmentum pontis in which the fillet was intact, but which were accompanied by anæsthesia of the opposite side. Added to this is the more important fact, which the experiments here recorded have brought out, that destruction of an entirely different region, viz., the posterior horn and gelatinous substance of Rolando, is followed by characteristic sensory disturbances on both sides of the body.

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[1] Kolliker, *Handb. d. Gewebelehre*, 1893.

[2] Held, *Arch. f. Anat. und Physiol. Anat. Abtheil.*, 1893.

[3] *Phil. Trans.*, vol. clxxxv., 1894, p. 763.

The experimental evidence in this paper is in favour of the view suggested by Tooth[1] on anatomical grounds, that the gelatinous substance and the adjacent part of the posterior horn are chiefly concerned in the transmission of sensory impressions.

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[1] Tooth, *Journal of Physiol.*, Supplement, 1892, p. 178.

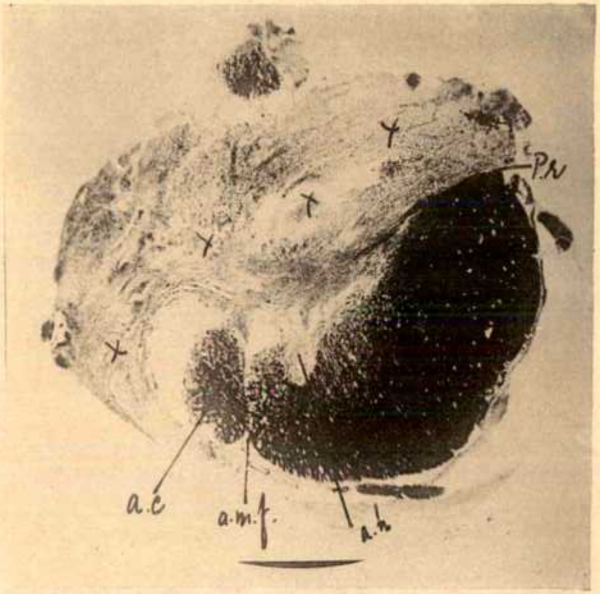
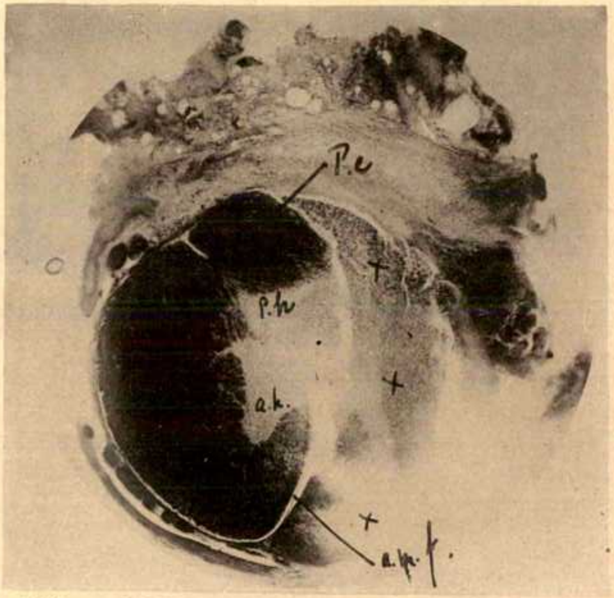


Fig. 5.



Collotype. J. J. Briginshaw, Sise Lane, 35d, Queen Victoria Street, E.C.

Fig. 6.

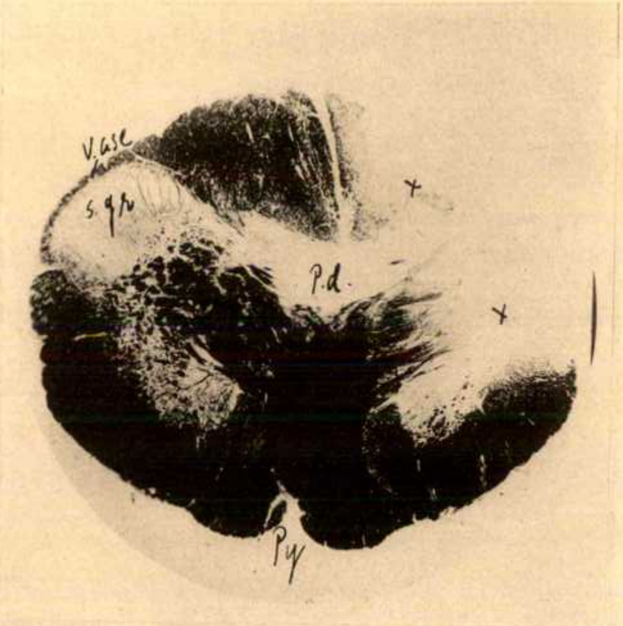


Fig. 3.

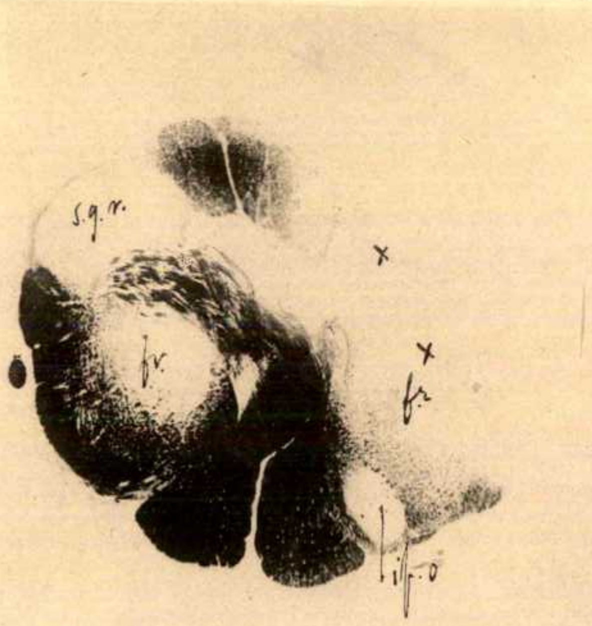


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Fig. 4.

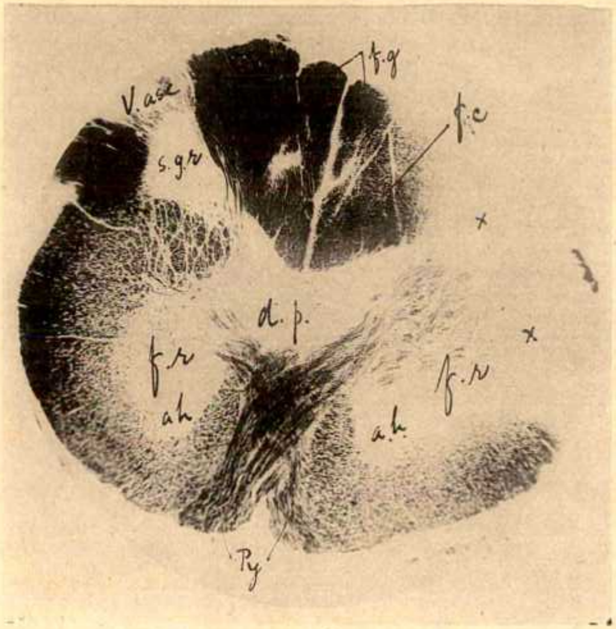

Fig. 1.

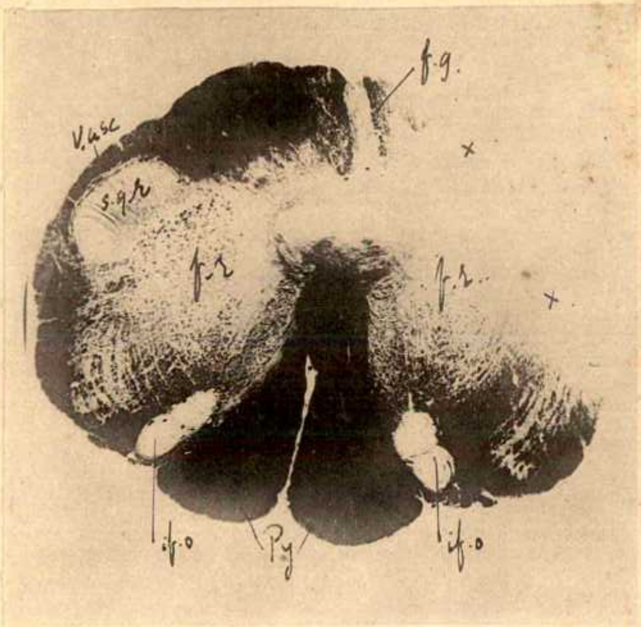

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Fig. 2.

EXPLANATION OF THE FIGURES.

FIGS. 1 and 2 illustrate the lesion in Experiment I.

xx. indicate the position of the lesion.
f.g. funiculus gracilis.
f.c. funiculus cuneatus.
s.g.r. gelatinous substance of Rolando.
a.h. anterior horn.
d.p. pyramidal decussation.
f.r. reticular formation.
if.o. inferior olivary body.
Py. pyramids.
V. asc. ascending root of trigeminus.

FIGS. 3 and 4 illustrate the lesion in Experiment IV.

xx. indicate the position of the lesion.
The lettering is the same as in figs. 1 and 2.

FIG. 5 is a section through the lesion from Experiment V. It will be seen that the whole of one side of the spinal cord, with the exception of the anterior column, is replaced by cicatricial tissue, which extends across to the posterior horn of the opposite side, this also being partially implicated.

FIG. 6 represents the extent of the lesion in Experiment VI.

a.h. anterior horn.
p.h. posterior horn.
P.c. posterior column.
a.m.f. anterior median fissure.
xxx. cicatrix.

Note.—In the figures representing the lesion of the Rolandic tubercle the outline of the cicatrix has not been depicted. This is due to the fact that the sections having been stained by the Weigert-Pal method, the fibrous tissue of the cicatrix was not sufficiently dense to retain any colour.

THE MORBID ANATOMY OF A CASE OF INFANTILE PARALYSIS.

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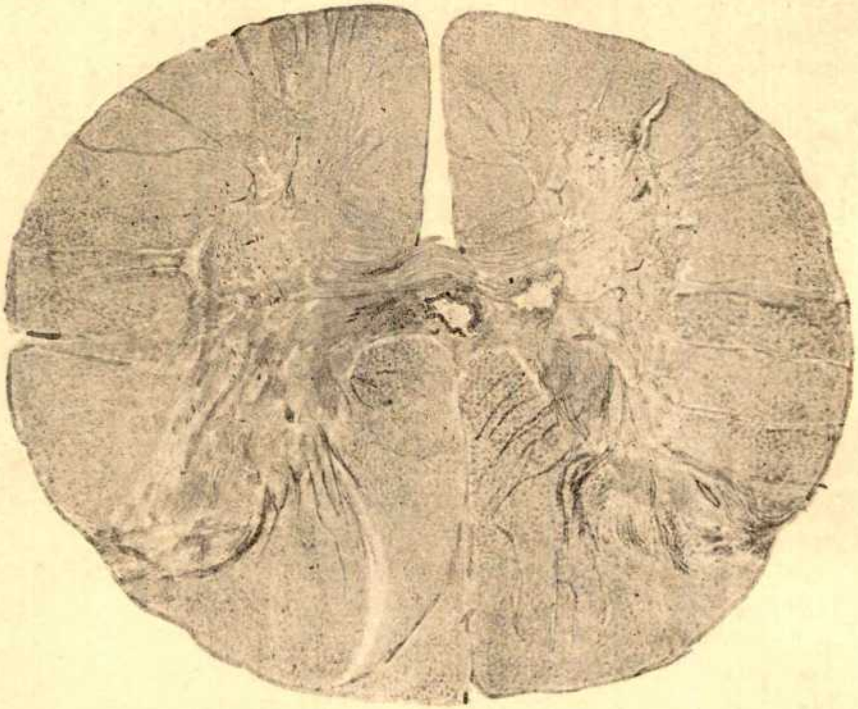
(*From the Laboratory of the Leeds General Infirmary.*)

THE following is an outline account of the case for the notes of which I am greatly indebted to Dr. Chadwick and Mr. Brown, under whose care the patient was.

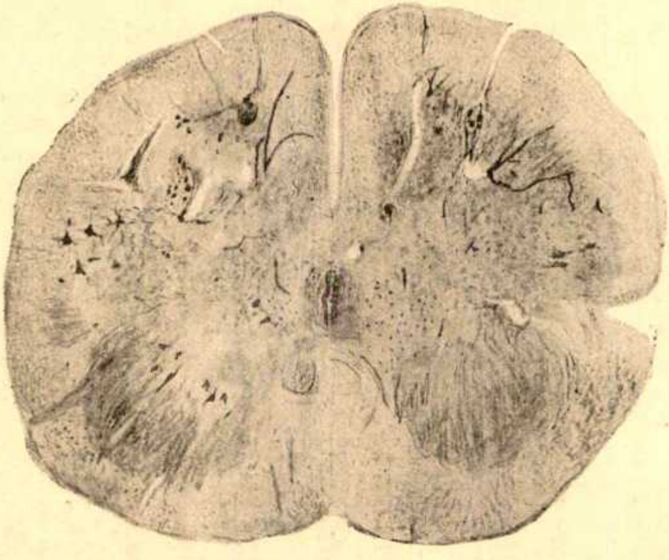
A girl, aged 6 years, was suddenly seized, nine months before admission, with fever, nausea and vomiting. Paralysis appeared on the second day, involving all four extremities. The fever ceased in nine days. The urine and fæces were passed into the bed for three weeks. The arms began to recover three weeks after the onset. On admission there was almost complete paraplegia with contracture in the calf muscles. The right arm was weak. Hardly any wasting was noted. The plantar and patellar reflexes were absent, the abdominal present. The reaction to faradism was absent, that to galvanism weak. K.C.C. = A.C.C. Sensation was intact. Later some improvement occurred in the legs, apparently under massage. Death took place about eleven months after the onset of the disease from independent causes.

At the necropsy there was slight diffuse nephritis with marked fatty changes in the glomeruli of the kidney. There was no obvious naked eye changes in the cord or brain. Unfortunately, I omitted to obtain any of the peripheral nerves or muscles.

A word may perhaps be said here in respect to methods. The rapid method of hardening in Müller's fluid in a hot oven at body temperature was adopted. In two and a-half



General View.—(*a*) Lumbar region. × 2 in.



General View.—(*b*) Lower sacral region. × 2 in.

days the material was taken out and put through alcohol of increasing strengths. In five days it was quite ready for embedding in paraffin. Sections were cut with the rocking microtome and were stained after Weigert's method, in aniline blue black, logwood and eosin, borax carmine or picro-carmine and logwood. A few sections were stained after Pal's method, and a few without much result in Rosin's triple stain.

Numerous sections were examined from both ends of the spinal nerve segments, longitudinal and sagittal sections through the horns being taken in the intervening parts. Sections were also examined from the M.O., pons, crura, internal capsule and motor cortex, but no lesions were found in these situations.

It may save space to summarise the morbid changes found in the various elements such as have been ascertained to exist in considerably over 200 stained specimens carefully examined and systematically noted.

Ganglion Cells.—The ordinary changes in these cells are readily seen. Alteration in and disappearance of the processes, as well as shrinking of the cell into an amorphous looking mass were observed. An indistinctness in the outline and a breaking up of the cell were occasionally seen. Only very exceptionally were round cells found in the periganglia space. Spaces where former ganglion cells had been were fairly numerous. A stippling of the general protoplasm of the cell was only obvious at times. There was no distinct evidence obtained of pigmentation or calcification of the cell. The changes in the nucleus and nucleolus were very interesting. It is often said that the nucleus and nucleolus disappear early, but there would appear to be circumstances under which these structures are more resistant to such changes. Shapeless masses of protoplasm were here sometimes recognised as the remains of ganglion cells solely by the presence of a distinct circular nucleus often with nucleolus obvious only on careful focussing. Some little time ago I was much struck when studying sections from a dog's cord with a spontaneous acute myelitis of five days' duration, to find that the nucleus of these cells could at times be seen almost shorn of all trace of surrounding protoplasm.

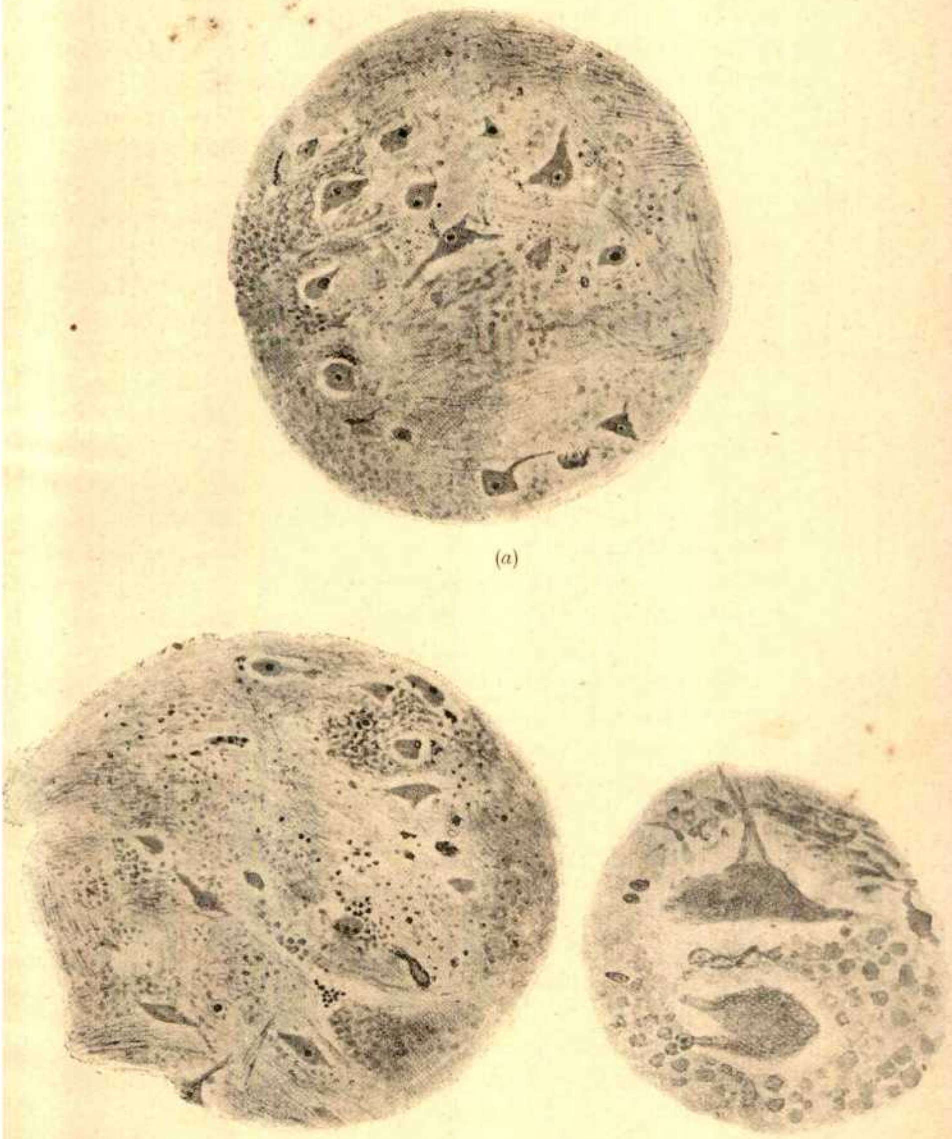
The above-named changes in the ganglion cells were best studied in the material from this case in the upper cervical and lower dorsal regions, and especially perhaps in Clarke's column (fig. 1). Groups of minute and perfectly formed ganglion cells were here and there seen chiefly in the parts corresponding to the lateral horns. Kohnstamm (*Zeitschr. f. klin. Med.*, 1893) has specially drawn attention to these cells. Once, and perhaps twice, a ganglion cell was seen to have two nuclei. In no part of the cord could the destruction of ganglion cells be said to be limited to one or more groups in the sense insisted upon by v. Kahlden.

The *nerve fibrils* in the grey matter were greatly changed. They had often entirely disappeared. When present they were mostly reduced to fine lines, and frequently they were distinctly varicose (fig. 2). These changes were readily studied in the anterior commissure. This fact is not so much to be wondered at if the median and hinder group of ganglion cells are to be looked upon as commissural cells (Lenhossek), for these latter were greatly involved throughout.

The *anterior nerve roots* as they passed out through the white matter seemed to be less well represented than usual, the individual fibres being fewer in numbers and thinner. Only in a few specimens did they appear to be degenerate. The posterior roots were intact.

The *neuroglia* of the grey matter was considerably altered, chiefly in the direction of overgrowth and infiltration. In many sections taken from the lumbar and cervical enlargements translucent areas were visible in the grey matter. These were unquestionably due to the destruction of the numerous nerve fibrils and ganglion cells. If such a translucent patch was examined under a high power, the overgrowth in the neuroglial element became obvious. It consisted of a fairly open rich network of thick fibres, with the presence here and there of enormous spider cells (fig. 3), whose processes could be distinctly seen to form this neuroglial network. In parts of the cervical region these

Fig. 1.



(*a*)

(*b*)

(*c*)

Ganglion cells healthy and in various stages of degeneration in Clarke's columns. The cellular infiltration is very well marked. Upper dorsal region.

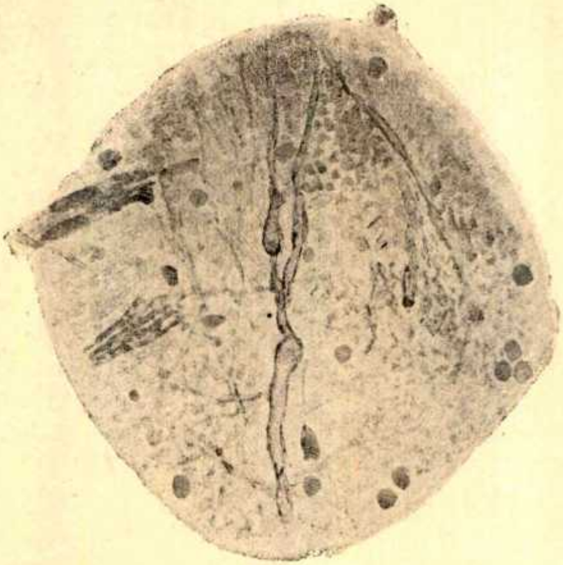


FIG. 2.

Varicose and wasted nerve fibrils from the upper lumbar region. × $\frac{1}{8}$.

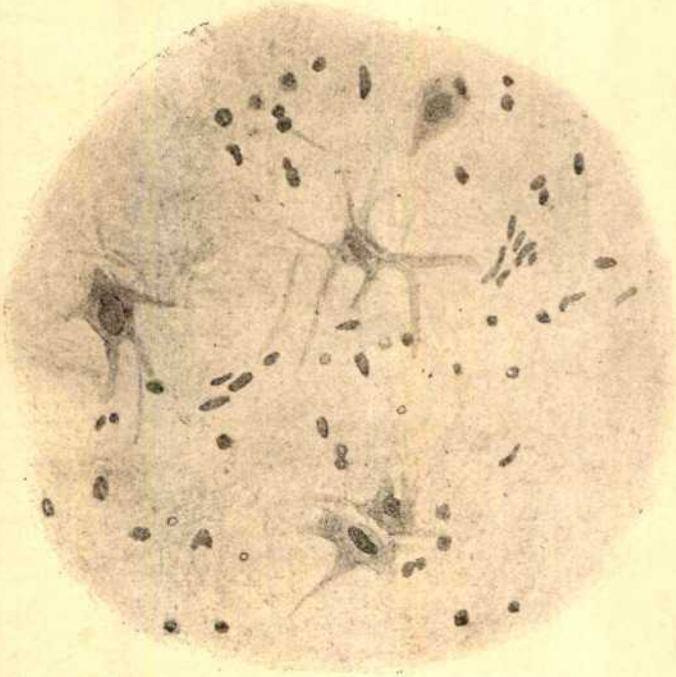


FIG. 3.

Enormous spider cells (lumbar enlargement). × $\frac{1}{8}$.

spider cells were present in great numbers. In Edinger's admirable *Vorlesungen*, 1893, the opinion is still expressed that these spider cells are really thin cell plates and that fibres only appear to arise out of the cells because they lie in close relation to them. That the fibres in question do arise from the cells in these specimens there can be no manner of doubt, and indeed, researches with Golgi's method seem to have set this question definitely at rest. (Lenhossek, *Der feinere bau des Nervensystems*, 1893.)

The *central canal* is pervious almost throughout the cord. In its neighbourhood are a number of large epithelial-looking cells clearly derived from the cells lining the central canal.

There are two further pathological processes in the neuroglia now to be considered, namely, the increased vascularity and cellular infiltration.

Vascularity.—In many parts of the cord there is obviously greatly increased vascularity. Here a few remarks may be made on the vascular supply of the cord especially as the vascular and interstitial origin of the disease is adopted here. It consists of central and peripheral components. The central artery penetrates into the anterior fissure at the bottom of which it divides. From this central supply Clarke's column is provided. These branches of the central artery can fairly readily be made out if a number of sections are examined.

The peripheral supply is chiefly limited to the white matter. It is very obvious, however, that the outermost parts of the grey matter also receive blood from the peripheral as well as the central arteries. In some of the sections such a branch can be seen to penetrate the anterior horn. As will be pointed out later, some of the advanced perivascular changes lie in a zone in which the central and peripheral arteries would appear to meet. Any distinct disease of the arterial wall itself I have not been able to make out except in a few doubtful instances. Once a capillary appeared varicose. The lymphatic sheath, however, was frequently found crowded with round cells.

Cellular Infiltration.—In most parts of the cord, and most marked in the mid-cervical region, there was very considerable cellular increase. These cells may roughly be divided, as has been done by Dauber and others, into (1) round cells with a very large nucleus, and (2) cells with many processes, namely Deiter's or spider cells. These latter would appear to vary very considerably in size. The cell infiltration is distinctly of two kinds: (1) patchy, (2) general. The patchy infiltration is frequently seen to be distinctly perivascular. The original specimen from which the drawing (fig. 4) comes, strangely reminds one of a recent infiltration. In another longitudinal section from the lumbar enlargement (fig. 5) the change would appear of a more chronic character, the neuroglia round these vessels being unquestionably considerably altered.

There is yet another infiltration, if it may be so called, round about the central canal and penetrating at times deeply into the surrounding parts. It consists of very large, often irregular, cells with no distinct processes. They have all the appearances of being derived from the cells lining the central canal. Since these cells have a similar origin to Deiter's cells, their increase and multiplication is hardly to be wondered at (see Lenhossek).

White Matter.—The changes here are much less obvious but yet distinctly present. In the neighbourhood of the diseased horns the neuroglial element is denser and its cells more numerous than usual. In one specimen an unmistakeable patch of round-celled infiltration was seen, and in another logwood eosin preparation a curious staining which looked exactly like the remains of an old hæmorrhage. No corpora amylacea were present. In longitudinal sections (logwood and eosin) the cellular increase could be distinctly made out. Similar sections stained according to Weigert's method showed slight changes in the myelin of the nerve fibres. In a few specimens in which the pia mater and subarachnoid tissue remained, no evidence of any inflammatory change was forthcoming. This is noted, as some observers seem to have found such a lesion present (Goldscheider).

As regards the distribution of the lesion, it extended undoubtedly throughout the whole length of the cord. It

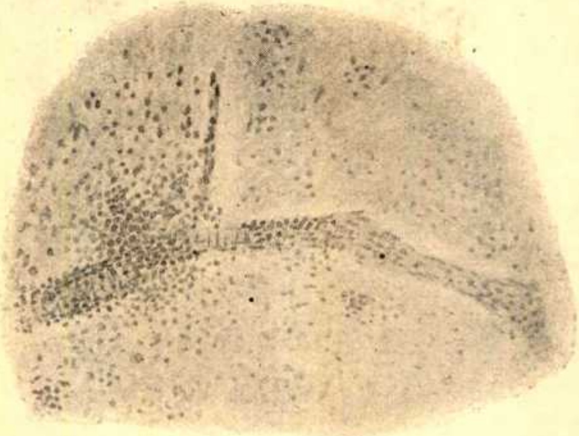


FIG. 4.
Perivascular infiltration (lumbar enlargement) about a vessel quite in the periphery of the grey matter. × $\frac{1}{8}$.

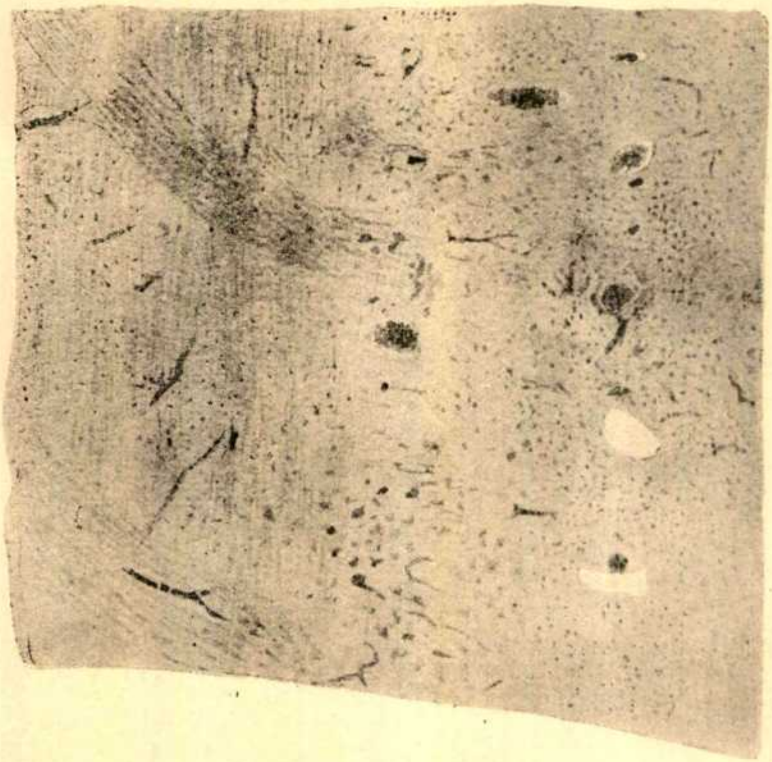


FIG. 5.
Longitudinal section, showing cellular infiltration and perivascular changes.

was present in the lowest sacral segments and reached as high as the decussation of the pyramids. The lesion was unquestionably intense in the *lowest* part of the cervical region (C6 to D1). It gradually diminished in an upward direction. It was also less intense in the upper dorsal region to become more marked in the mid-dorsal region and less pronounced in the lower dorsal cord. In the lumbar region it was very intense, especially about L 2, 3 and 4. Below L 5 it tended to become less marked, but did not altogether disappear even in the lowest sacral segments. It was somewhat less marked, especially in the cervical enlargement, on one side than on the other.

It is proposed briefly to attempt an explanation, however inadequate, of the clinical features of the case, by the morbid lesions found.

The paralysis in the lower limbs is amply accounted for by the disease in the lumbar cord. The improvement noted in the legs in the tenth or eleventh month, so that the child could just lift them from the bed, is curious. In one longitudinal section, however, from the upper lumbar enlargement quite a number of healthy ganglion cells were found. In no other section from this region was a similar appearance noted. It is almost surprising that there was any power at all in the right arm, and yet the patient could move the fingers of the corresponding hand. The amount of power possessed by the left arm is also remarkable, for the child could feed itself with this left arm. It would be a hardihood to attempt to explain the above noted improvement by a regeneration of ganglion cells. Yet those small and perfect ganglion cells noted above suggested such a possibility. Tumours have been found in the central nervous system composed in great measure of ganglion cells, showing that the division and multiplication of these cells is sometimes possible.

The absence of wasting was a striking fact; it was noted clinically, and in the pathological report. The absence of the plantar reflex could sufficiently be accounted for by the disease in the sacral segments, and the absent knee jerk by the intense disease in the lumbar enlargement. The abdominal reflex was present, and the disease was certainly less intense in the corresponding part of the cord (lower dorsal). Unfortunately there is no note about the other reflexes. The epigastric reflex should have been absent on account of the marked lesion in the mid-dorsal region.

The question of the nature of the lesion in infantile paralysis has been so fully debated of recent times, and the disease so clearly shown to be of vascular and interstitial origin—that is, the changes in the neuroglia including vessels, are primary, those in the nerve cells secondary—that it is unnecessary to discuss it at length. Goldscheider (*Zeitschr. f. klin. Med.*, 1893) and even more specially Siemerling (*Arch. f. Psychiatrie*, xxvi. Bd. i., 1894) have strongly advocated this view. v. Kahlden (*Ziegler's Beiträge*, 1893) has, however, vigorously supported Charcot's opinion that the disease starts in the nerve cells, the changes in the neuroglia being secondary only. Siemerling's rejoinder seemed unanswerable. v. Kahlden has again quite recently returned to the subject in the *Centralbl. f. allg. Pathol.*, Sept., 1894 (Epitome, *Brit. Med. Jour.*, Oct. 20, 1894, par. 292) in a review of recent writings upon this question. He discounts, perhaps hardly pertinently, some of the cases which have been brought forward in favour of the interstitial and vascular origin of the disease, by treating them as atypical cases.

Most of the cases upon which the above authors have based their conclusions are of very recent origin. In the old cases the changes are so advanced as almost to preclude an opinion as to the starting point of the disease. The case recorded above stands in a middle group between these two classes of cases. The changes seem to be entirely in favour of the disease being primary in the neuroglia including the arteries. The infiltration, and especially its perivascular localisation, may be here especially referred to. On the other hand, there is no definite localisation of the disease to any one or more groups of ganglion cells. The overgrowth in the neuroglia, including the spider cells, is certainly striking. This may be due to the irritation of the poisonous products (toxines), escaping from the blood according to the infective theory of the disease, and this view certainly appears to offer the best explanation of the etiology of the disease. In more chronic cases the overgrowth of the neuroglia might be looked upon to some extent as supplementary (*vacatswucherung*), that is, the permanent vascularity being certainly not diminished and often increased, whereas the nerve elements have perished, there remains less tissue to nourish, hence the increased nourishment to the neuroglia leads to its overgrowth. Changes have been found in the neuroglia in quite recent cases, so that the irritation theory would appear best to account for the changes in the neuroglia, certainly in the earlier cases. In respect to etiology, it is interesting to note that micro-organisms have not been found in the cord. Yet the possible occurrence of epidemics of infantile paralysis as recorded by Medin (*Centrabl. f. klin. Med.*, 1891), and the experimental evidence as given by Roger (*Rev. de Méd.*, 1893), and more recently by Thoinet and Masselin (*Rev. de Méd.*, June, 1894), strongly support the view that the disease is an infective one. Thoinet and Masselin injected preparations of the B. coli communis and Staphylococcus, and determined spinal lesions at varying intervals after the inoculation, the peripheral nerves remaining intact. It is the poisons excreted by the micro-organisms that set up the disease, and this accounts for the early vascular phenomena found in the cord.

ON ENDOTHELIOMA OF THE SPINAL DURA MATER: WITH A CASE IN WHICH AN OPERATION WAS PERFORMED.

BY J. MICHELL CLARKE, M.A., M.D., M.R.C.P.

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THE following case of endothelioma of the spinal dura mater presents several features of both clinical and pathological interest:—

A. P., aged 28, a bootmaker, came to the hospital on April 1, 1892, complaining of loss of power in the arms and legs. He had never had any previous illness. Eight years ago he had a sore on the penis, which was not followed by a rash, sore throat, or any other symptoms. In July, 1890, he had an attack of shivering, followed by fever, loss of appetite, and vomiting. There were looseness of the bowels and delirium; but after a few days he returned to work well. In July, 1891, he had a fall from his bicycle on to his left side, but apparently sustained no injury. When riding his bicycle in the early part of this year, he occasionally felt numb sensations in the legs. In September, 1891, the present illness began with twitching sensations in the hands on waking in the morning, passing off after breakfast. He also noticed a difficulty in holding small objects, and that when he had a tool in his hand he could not at once put it down. All these symptoms gradually grew worse, and the legs began to grow weak, until he had to give up work on December 3, 1891. Besides weakness of legs and arms, the muscles of the latter slowly wasted. With the exception of some dull aching pain in the shoulders, the loss of power was not accompanied by pain, and there were no shooting pains in either arms or legs. There appears to have been loss of sensation at this time, for he several times, whilst lighting the fire, burnt his left hand without being aware of it. He had had no affection of sight or of speech, no attacks of sickness, no difficulty in breathing, micturition, or defæcation. The wasting of the arm muscles began later than the loss of power and sensory affections. He was positive as to the absence of severe pain throughout the illness and of injury to the spine.

Symptoms on Admission, April, 1892.—Patient was a tall, well-nourished man, of good general health. He spoke distinctly. There was no oculo-motor or facial paralysis. He walked badly, with an unsteady gait, dragging the left leg, which was kept stiff and rigid; he was unable to turn round sharply. He could not stand with his feet together and eyes closed. When lying down he could only lift the right foot with difficulty; the left hardly at all. He had great difficulty in picking up a pin with either hand, especially with the left, and he could not button his clothes. The fingers were flexed at the last two phalangeal joints. *Right hand:*—Movements feeble, especially in extensor indicis. *Left hand:*—Interossei and abductor indicis paralysed and somewhat wasted; slight atrophy of muscles of thenar eminence, with tendency of thumb to lie in ape-like position. The reaction of these muscles to faradism was feeble. He could bring his right hand up to his mouth, but not the left. The muscles of the left arm and forearm were smaller than those of the right. There was loss of sensibility over each forearm and outer part of each arm. The legs showed well-marked rigidity. The knee-jerks were exaggerated; ankle clonus present on each side, spontaneous, clonic spasm of legs occurred at intervals on standing. The abdominal and cremasteric reflexes were at this time not obtained. No loss of sensation over lower extremities, nor over chest, but there was some deficient sensation over upper part of abdomen. Ten days after admission, whilst walking in the ward, he slipped and fell on his face; after the fall he became much worse, lost all power of walking or standing, and nearly all movement in his arms. Except for some aching and burning sensations in the limbs, which lasted only a short time, he had no pain after the fall.

May 1, 1892.—He could raise the shoulders and arms slightly, but could not depress the raised arm, nor lift the arms off the bed, and had absolutely no movement in the fingers, wrists, and forearms. There was complete loss of power in the legs, which lay in position of extension. The arms lay by his side, with the elbows flexed, the forearms across the epigastrium on their palmar aspect, the wrists flaccid, neither flexed nor extended, and the fingers flexed at the phalangeal joints. All the joints could be passively flexed and extended. The muscles of the back were decidedly weak.

There was marked clasp-knife rigidity of legs, with knee and ankle clonus. Plantar reflexes feeble—lower abdominal present, upper abdominal absent. No wrist-jerk; doubtful whether a feeble elbow-jerk was obtained.

Sensation was lost to all forms over both hands, except that a pinch could just be felt in the palms, and sensation to firm pressure was present over the backs of the second and third fingers of the right hand. Touch, pain, and temperature sense absent over backs of forearms; over palmar surface of forearms very deficient sensation to touch and pain; temperature sense preserved. The contact of a moderately hot test-tube with the back of the right arm raised a blister, which healed very slowly. Over the arms the temperature sense was absent; touch and pain were almost normally felt. Sensation was deficient on the chest from the second to the fifth ribs, and lost over an encircling band extending behind from the fifth to the eighth dorsal spines, from the spine of the scapula to one inch below its lower angle, and in front from the fifth to the level of the tenth rib. There was no loss of sensation over the head and neck, nor over the lower extremities. The bladder and rectum remained unaffected.

Three weeks later, a peculiar modification of sensory disturbance, resembling that found in syringomyelia, was observed over the arms and over the chest, from the clavicles to the level of the lower border of the pectoralis; on this area there was loss of sensation to pain and temperature, but complete preservation of the tactile sense, so that two points could be distinguished at the normal distance and the light contact of a single hair be detected. The sense of position was unaffected.

In July, 1892, the condition was much the same, with the exception that the wasting in the muscles of the upper extremity had certainly increased. Fibrillary tremor was present in the wasted muscles; those chiefly affected were the deltoids and small muscles of the hands, especially of the left hand. These muscles showed greatly diminished reaction to faradic and increased to galvanic currents. The other muscles of the shoulders, arms, and forearms were wasted, but to a less extent. He complained of burning and tingling pains under the finger-nails and at the finger-tips. Sensation same, with the peculiar disturbance over the limited area mentioned above. The diagnosis made at this time was that of pachymeningitis in the cervical region, with pressure on the cord, possibly of syphilitic origin. It was recognised, however, that the symptoms did not exactly correspond to those of pachymeningitis cervicalis hypertrophica, especially in the absence of severe pain. The sensory modifications over the arms and chest suggested syringomyelia. He was treated with full doses of iodide of potassium, with mercurial inunction and counter-irritation over the cervical spine, but without any benefit, and after being in the hospital about ten months, as the disease seemed stationary, and no further treatment was at that time contemplated, he was discharged.

He returned to the hospital in July, 1894, with an urgent desire that something more should be done for him. Some friend had told him of the possibility of an operation affording him relief, and he wished that anything should be tried which would give him any chance of some recovery of power, if only the ability to feed himself.

Condition on Re-admission.—He was perfectly helpless. He lay on his back with his legs drawn up, and any stimulus such as withdrawing the bedclothes caused them to be still further drawn up. The right arm was slightly abducted, the elbow flexed, the forearm pronated and lying across the chest, the wrist was somewhat extended and the fingers flexed into the palm; the thumb was slightly flexed and adducted lying outside the fingers (modified "main en griffe"). The fingers and thumb could only be passively extended by using some force. Both extensor and flexor muscles were wasted; the latter the most so. The small muscles of the hand showed the greatest degree of wasting. The patient could accomplish very slight flexion, extension, and pronation of the forearm, and could raise the shoulder directly upwards to a small extent, but all these movements were very limited and of no practical service to him.

The left upper extremity was kept extended by his side, the forearm being prone. The fingers and wrist lay in the same position as on the right side; he had no power of movement in this limb. The legs were rigid, the muscles being hard, and not wasted; the patient could slightly flex them at the hip and knee-joints, but had no power over the ankles or toes. The leg muscles reacted to both faradic and galvanic currents. The head and neck could be moved freely in any direction. There was no paralysis of any cranial nerve. He had no control over the bladder, and suffered from obstinate constipation. The finger-nails showed trophic changes, being white and brittle.

Reflexes.—Abdominal, plantar and cremasteric all increased. Knee-jerks exaggerated, especially on the right side; patellar and ankle-clonus easily obtained.

Electrical Reactions.—Faradic current: right arm. No reaction in deltoid; long flexors react very feebly; no reaction in small muscles of hand; other muscles react well; biceps reacted better than triceps. Left arm.—No reaction obtained for pectoralis major, deltoid, biceps and intrinsic muscles of hand; other muscles contract.

Right Arm.

Constant Current.

Biceps	KCC = 3 milliampères	ACC = 5 ma.
Pectoralis	KCC = 20 ma.	
Deltoid	KCC = 10 ma.	ACC = 9 ma.
Triceps	None with Kathode	ACC = 15 ma.
Long extensors of fingers	KCC = 2 ma.	ACC = 5 ma.
,, flexors ,,	No reaction	ACC = 12 ma.
Dorsal interossei	KCC = 2·5 ma.	ACC = 5 ma.
Thumb muscles	No reaction.	

Left Arm.

Constant Current.

Biceps	No reaction up to 20 ma. of current.	
Pectoralis	KCC = 10 ma.	ACC = 0.
Deltoid	No reaction.	
Long extensors of fingers	KCC = 4 ma.	ACC = 4.5 ma.
,, flexors ,,	KCC = 15 ma.	ACC = 15 ma.
Dorsal interossei	KCC = 12 ma	ACC = 12 ma.
Thumb muscles	No reaction.	

From the accompanying charts the disturbances of sensation present at this period of the case can be seen. The peculiar retention of tactile with loss of pain and temperature sense over a certain area observed earlier in the case was now absent. On this account several careful investigations were made of the state of sensation to heat and cold, the results of which are given in figs. C and D, which show curious anomalies of the temperature sense over the body surface. The diagnosis at this time seemed on the whole to be in favour of a swelling external to the cord and pressing upon it. As there was no sign of disease of vertebræ, this was not likely to be a mass of caseous tubercular material, such as is found compressing the cord in tubercular caries. The absence of any improvement under treatment by iodide of potassium, and of positive evidence of syphilis, rendered a gumma or syphilitic inflammatory swelling of the membranes improbable. The duration of the illness, and the fact that during the last eighteen months the paralysis of sensation had remained stationary, whilst in the case of the motor paralysis there had been slight improvement in the arms, negatived the existence of tumour of a malignant character. It was considered that the sudden increase of paralysis after a fall during his previous stay in the hospital was due to a patch of myelitis at the seat of lesion.

Both motor and sensory symptoms indicated that the lesion lay from the origin of the fifth to the eighth cervical nerve roots. The symptoms pointed to considerable vertical extent of the lesion of the cord, and on this account the probability of a growth seated centrally within the cord had to be taken into consideration, but the long duration and stationary character of symptoms precluded a tumour of this kind, which is generally a glioma. Considerable vertical extent of lesion is also found in cases of meningitis, and the cervical region of the cord is the common seat of hypertrophic pachymeningitis, with the symptoms of which those of my patient corresponded fairly well, but for the important exception of absence of the severe radiating pains generally present in this disease.

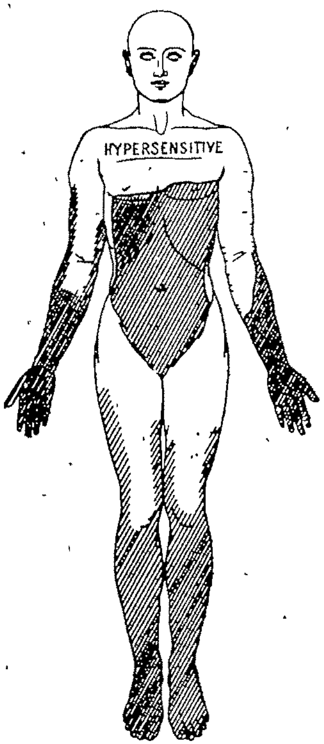


FIG. A.

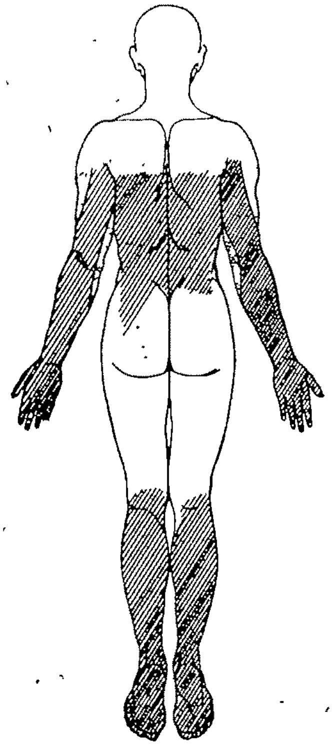


FIG. B.

An exact diagnosis of the nature of the disease was thus unfortunately not arrived at, but the condition was considered to be a benign tumour of uncertain nature, or a localised thickening, probably seated in or outside the spinal membranes, and compressing the cord. Dr. J. A. Ormerod, who was in Bristol for the July meeting of the British Medical Association, 1894, kindly saw the case with me, and concurred in this view. The patient, who

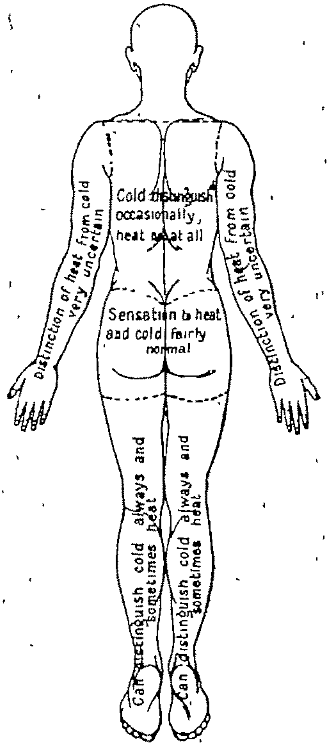

Distinction of heat from cold very uncertain
Cold distinguish occasionally, heat not at all
Distinction of heat from cold very uncertain
Sensation to heat and cold fairly normal
Can distinguish cold always and sometimes heat
Can distinguish cold always and sometimes heat

FIG. C

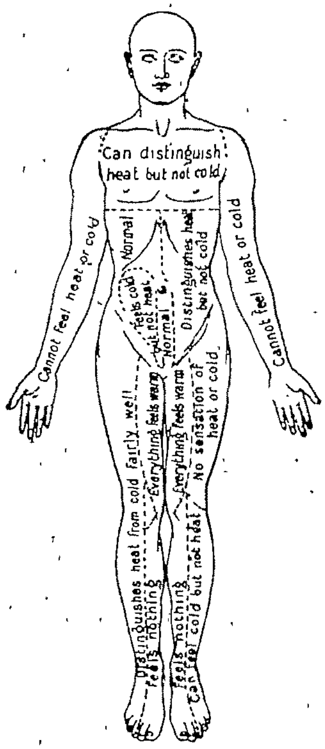

Can distinguish heat but not cold
Cannot feel heat or cold
Normal
Feels cold but not heat
Normal
Distinguishes heat but not cold
Cannot feel heat or cold
Distinguishes heat from cold fairly well
Everything feels warm
Everything feels warm
No sensation of heat or cold
Feels nothing
Feels nothing
Can feel cold but not heat

FIG. D.

was a very intelligent man, was very anxious that something should be done for him, and the dangers of an operation and the possibility that no good might result from it were put plainly before him. I decided to wait a month, at any rate, until I returned from my holiday, and that he should fully consider the question in the meantime. The fact that he had regained some slight power in the right arm and that most muscles in the arms still retained some electrical irritability, led me to hope that if the pressure could be removed, some recovery in the arms might ensue, even after the long duration of symptoms, and that the spasmodic contractions of the leg muscles, which gave him much pain, would be relieved. On my return, he still strongly wished that an operation should be attempted, and my colleague, Mr. W. M. Barclay, accordingly operated on September 12. He has kindly furnished me with the following notes:—

"On September 12 I explored the cervical region of the cord. The patient was anæsthetised with chloroform, and placed in the semiprone position, and an incision was made over the spines from the fourth or fifth cervical to the first dorsal—this being subsequently extended a little downwards. The muscles were rapidly detached from the spines and laminæ, first on one side and then on the other, by the knife—the vertebral aponeurosis being transversely divided on each side to allow of freer retraction of the muscles, as recommended by Mr. Horsley. The bleeding was very excessive and very difficult to stop, and the patient lost a great deal of blood before this was accomplished. The sixth and seventh cervical spines were then cut off by bone forceps, and the seventh laminal arch trephined—the arches of the sixth, fifth, and fourth being subsequently removed by forceps. On thus exposing the spinal canal, it was found to be completely filled with a reddish-grey, smooth material looking like the reddened dura mater. When, however, I incised the supposed dura mater, I cut into a greyish granular and gelatinous-looking substance, which proved to be entirely outside the dura; it extended as far up as the fourth lamina, possibly further, and dipped under the first dorsal arch below; on each side it passed forwards round the cord among the nerve roots, and could not be further exposed. The patient had been getting very faint for some time, and at this stage he became so collapsed that I was obliged hastily to cut away a small part of the mass on the dorsal aspect and close the wound. With warmth and injections of brandy, hypodermically and *per rectum*, he seemed to rally somewhat, but the collapse returned, and he sank the same night.

"The excessive loss of blood at the early stage of the operation was most untoward, and unlike any I had previously seen in clearing the spine. It seemed unavoidable, and I cannot see what measures could have been adopted with a better chance of success. A still further delay was unfortunately caused by the forceps (Victor Horsley's laminectomy forceps) being unable to bite through the laminæ. In consequence of this I had to use ordinary bone forceps, which are very clumsy for this work.

"I had hoped (had he survived the operation) that by removing the dorsal part of the mass around his cord, the pressure might be, to some extent, relieved, and the patient's condition ameliorated."

The autopsy was made seventeen hours after death. There was the scar of an old bed-sore over the sacrum. The operation wound in back of the neck looked healthy. The posterior arches of the fourth, fifth, sixth, and seventh cervical vertebræ had been cut away, and the dura mater lay exposed in the floor of the wound, in which lay a little blood. On laying open the spinal canal, dura mater and cord appeared quite normal under the wound, except for a minute fragment of pale growth immediately below it. Above the wound the cord was covered posteriorly by a flattened layer of somewhat friable new growth, which extended around it forwards so as almost to enclose it; the growth lay outside the dura mater, and was adherent to the inner surface of the posterior arches of the third and fourth cervical vertebræ. The membranes were adherent to each other and to the cord over the entire extent of the tumour, and the cord was also very soft for a corresponding distance, and was decidedly flattened from before backwards. Owing to its softness and to the surrounding adhesions, it was difficult to remove, and was slightly torn. The growth did not extend on to anterior surface of cord at level of operation wound, but did so slightly above it. The cord, which, to the naked eye, in the fresh state, showed no degenerated tracts, was at once placed in Müller's fluid. The vertebræ were everywhere normal in all respects; there was no sign of caries, of old fracture, or growth in them, and the tumour was not connected with them.

On further examination after hardening, the tumour was found to extend from the origin of the fifth cervical nerve roots to that of the first dorsal. It lay chiefly in the posterior aspect of the cord, which it had flattened and compressed throughout nearly the whole of the cervical enlargement. Its greatest bulk was on the left side of the posterior aspect of the cord; the nerve roots were somewhat compressed and atrophied from pressure by the tumour, perhaps especially on the left side; but all those corresponding to the vertical extent of the tumour were more or less affected. It lay external to the dura mater, appeared to be loosely attached to it, leaving a rough surface where removed. There was nowhere any trace of hæmorrhage into the tumour, which was soft, and not tough, nor was it laminated in structure. It was about 2¼ inches long, of which a piece, about 1 inch in length, was removed at the operation.

Its breadth on removal, extending round cord, was 1¼ inches; its average thickness ¼ inch, greatest thickness, ⅜ inch. The membranes under the growth were adherent to each other to the cord.

Circumference of spinal cord ⅜ inch above upper edge of growth..	2¼ inches
,, ,, ,, ,, at middle of growth about middle of cervical enlargement..	1¼ inches
Circumference of spinal cord, end of cervical enlargement . ..	1⅞ inches

These measurements show the extent of the compression of the cord.

Sections of the cord were stained by Weigert's method, and with carmine and logwood.

Throughout a vertical extent in correspondence with that of the growth, the cord had undergone extensive destruction from pressure, and at the point of greatest softening its different structures were distinguished with difficulty; but except for a very short distance the main features of its structure were clearly recognisable. It was noticed that although both grey and white matter had undergone extensive destruction, in sections stained by Weigert's method there were found many fibres in the white columns which had retained their myelin sheaths. These fibres were fairly numerous in both the posterior and anterior, and lateral columns, but especially in the latter and were present in all sections, including those from the part which had suffered greatest compression.

Weigert's stain also brought into view ascending degenerations, chiefly in the posterior columns, above the tumour, and descending degeneration in the pyramidal tracts below it, although these were not visible in the fresh cord. Neither in the ascending and descending tracts was the degeneration complete, in all of them about a third at a rough estimate of the total number of fibres still retained their myelin sheaths and were to all appearance healthy. There was no growth within the membranes or within the spinal cord itself. In the upper part of the cervical region one or two longitudinal slits were present in the posterior part of the cord near the base of the posterior horn; but careful examination showed that these were produced during the removal of the cord.

Portions of the tumour were embedded in celloidin and sections stained, some in logwood and resin, and others in picro-carmine.

Microscopically the tumour was seen to spring from the outer surface of the dura mater; its structure is represented in the figures. It belonged to the endotheliomata or psammomata, more correctly speaking to the former, as there were no calcareous granules in it. The stroma was composed of rounded or oval nucleated cells lying in a partly structureless, partly indistinctly fibrillated matrix. Passing through the tumour were vessels and also trabeculæ. In some parts the cells had a roughly alveolar or lobular arrangement, apparently determined by the course of the vessels. Scattered through the tumour were endothelial cell nests. The individual cells composing these could not be made out, save in one or two places where they were in process of formation. These nests seemed to be of slow growth, if one may judge from a comparison of their structure with the richly nucleated endothelial cell nests in a case of psammoma of the cerebral dura mater under my care (see figs. 4 and 5). Outside the endothelium of the small vessels was a layer of homogeneous hyaline-looking material, in the position of the muscular coat, but thicker than a normal muscular coat, and also present in the smallest vessels. This stained pink with picro-carmine, as did also some bands of similar appearance which passed through the tumour and did not contain vessels. The cell nests show the same hyaline material in their middle and external layers. Ernst, in a paper on psammoma ("Ueber Psammoma," *Beiträg. z. Path. Anat. und z. Allgemein. Path.*, 1891-2, p. 234), describes and figures the band of hyaline material which forms in place of muscle a hyaline layer outside the endothelium, and also thicker bands of the same substance running through the tumour. He regards the hyaline substance as a change antecedent to calcification. This paper gives an excellent account of the best methods of staining and of various questions as to the nature and genesis of psammoma.

This hyaline change in vessels and cylinder-like bodies is also described by Drs. Bremer and Carson ("Cylindroma

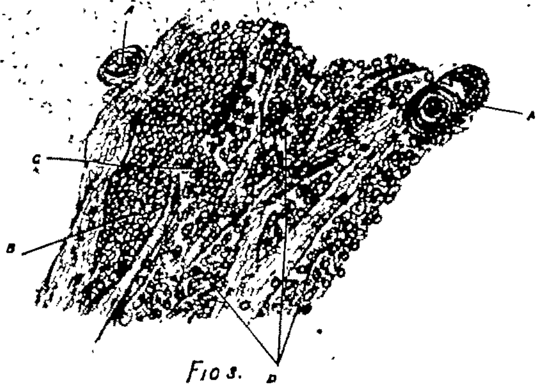
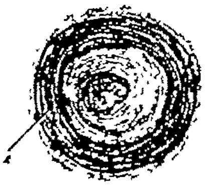


Fig 3.

Shows Spaces formed by Trabeculæ of Similar Structure to Vessel Walls containing Cells of Stroma and Numerous Red Blood Corpuscles.

A A—Cell nests B—Vessel with narrow imperfect wall on one side through which blood corpuscles are escaping at C. D—Spaces containing numerous blood cells.—*Zeiss*, Apochrom. 4 mm. Compens. Oc. 4.



Cell Nest more Highly Magnified.

A—Layer resembling in appearance wall of vessel.

Fig 4.

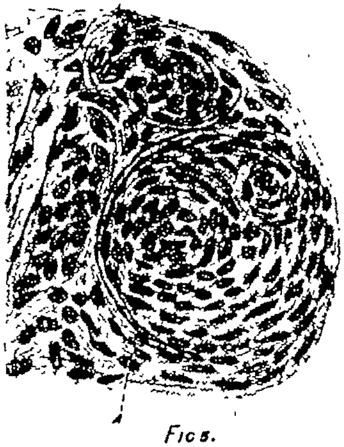


Fig 5.

Cell Nest from Case of Psammoma of Cerebral Dura Mater.

Comparison of figures shows richness of latter in nuclei. Cell nests in former smaller, and neither with logwood nor picro-carmine were nuclei brought out; with logwood nests appeared homogeneous in outer layers, staining a very intense blue. Cell nest in fig. 4 is thus of slower growth than that in fig. 5.—*Zeiss*, Apochrom. 4 mm. Compens. Oc. 8.

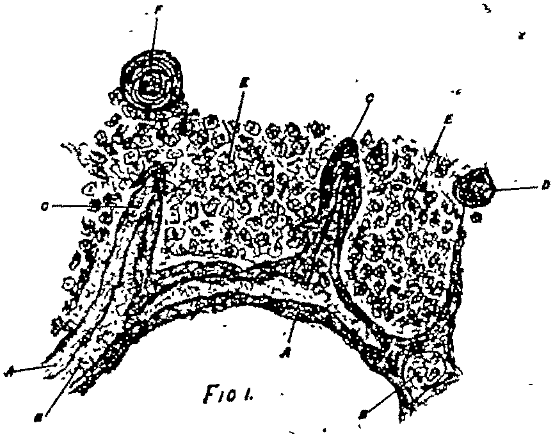


FIG 1.

VESSEL WITH HYALINE HOMOGENEOUS WALLS.

A-A—Vessel wall. B B—Lumen with blood corpuscles. C C—Buds given off from vessel. D—Small cell nest of similar hyaline substance apparently attached to vessel by slender thread. E E—Cells of stroma. F—Cell nest.—*Zeiss*, Apochrom. 4 mm. Compens. Oc. 4.

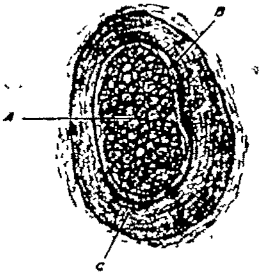


FIG 2.

VESSEL MORE HIGHLY MAGNIFIED.—*Zeiss*, Apoch. 4 mm. Compens. Oc. 8.

A—Lumen of vessel. B—Endothelial layer (? broader than normal).
C—Hyaline coat of vessel.

Endothelioides of Dura Mater," *Amer. J. Med. Sci.*, Feb., 1895, p. 120), and they also figure as "capillary angioma" in fig. 4 of their paper much the same appearances that I have drawn in fig. 3. The walls of these blood spaces appear to consist of pink stained homogeneous hyaline substance. As to staining I may briefly mention that picro-carmine gave very good results for the vessels and cell nests; logwood stained the cells of the stroma well, but the cell nests were inconspicuous in logwood stained sections, and their true nature could only imperfectly be distinguished. I made my sections before reading Ernst's paper, and could not follow the method there recommended and quoted from Van Gieson (*N.Y. Med. Journ.*, July 20, 1889) of staining with a mixture of acid fuchsin and picric acid and for nuclei of cells with Dalafield's hæmatoxylin. Picro-carmine gave, however, very good results indeed, especially when supplemented for cells of tumour-stroma by a comparison of sections stained in logwood.

Endothelioma, or psammoma, seems to be relatively of more seldom occurrence in the spinal than in the cerebral meninges. No doubt some of the cases described in the older writings on tumours of the spinal meninges as "fungus of the dura mater," epithelioma and fibroma of the dura mater include examples of the different forms of psammoma or endothelioma. Wilks and Moxon mention ("Pathological Anatomy," p. 249), without further particulars, having met with one case of psammoma of the spinal membranes, and I have found the following cases in a fairly extensive search through the literature. It seemed, however, useless to go very far back, as the microscopic structure of tumours of older date was not clearly differentiated.

(1) J. Hutchinson, Junr. (*Path. Trans.*, vol. xxxiii., p. 23): Woman, aged 48, tumour 1¼ in. by ½ in. in dorsal region of cord; microscopic calcification had occurred in it. Duration of symptoms two years; paraplegia without loss of sensation; very severe and constant pains shooting into lower part of abdomen and down legs.

(2) Mr. J. Hutchinson refers to a case described by Dr. Wilks (*Path. Trans.*, vol. vii., p. 37) as a tumour of fibro-

nucleated structure, not simple fibrous growth, in a woman aged 43. Tumour size of a bean, of oval shape, firm, hard, and grey, and growing from dura-arachnoid. Symptoms of nine months' duration, began with pains between shoulders and in chest and legs. She suffered from weakness of legs, but without complete paralysis and without sensory affection, and from violent pains and convulsive twitchings in limbs and abdomen. Dr. Wilks adds to the account of this case that: "In the year 1849 a man died in Guy's Hospital from a very similar tumour of exact form and size, and growing from the like part of the cord."

(3) H. A. Lediard (*Ibid.*, vol. xxxiii., p. 27): Tumour of dura mater pressing upon cord in cervical region. A note added to description says that specimen is in museum Royal College of Surgeons described as endothelioma connected with pia mater of spinal cord. Man, aged 25: duration of symptoms seventeen months. Pain and stiffness in neck; numbness, loss of power in left upper extremity; right arm next involved in same way, until finally there was complete paralysis of arms, legs and trunk. Death from implication of muscles of respiration. Tumour smooth, firm, pinky-white, irregularly lobed. Length ⅞in. by ⅝in. broad by ⅜in. thick.

(4) Dr. William Cayley (*Ibid.*, vol. xvi.): Oval tumour at interval between tenth and eleventh dorsal vertebræ growing from inner surface of dura mater, not adherent to cord; tumour soft, and white in colour. Woman, aged 46: duration of symptoms twelve months; 1¼in. long, by 5 lines thick. Calcified cells in centre of nests.

(5) T. Whipham (*Ibid.*, vol. xxiv., p. 15): Tumour opposite sixth and seventh cervical vertebræ, grew from dura mater, and was firmly adherent to it; though lying in such close proximity with cord, had no connection with it. The patient was a woman, aged 34: duration of symptoms, which began in legs, thirteen months. There is also a case reported by Dr. Ogle as carcinomatous tumour of foramen magnum (*Trans. Path. Soc.*, 1852-53), but no account of the microscopic structure of the growth is given.

Although the number of recorded cases of psammoma, or endothelioma of the cerebral meninges is very large, the above are all the cases of growths in the spinal membranes that were certainly of this nature that I have been able to find in the literature accessible to me. I have, however, been unable to peruse two or three papers, especially one referred to by Ernst ("Ueber die primitiv geschwülste der Dura Mater," *Wein. Med. Jahrbucher*, 1874), which is based on twenty-eight cases, and one by MacAlister, "Sarcoma Spin. Meninges," Zurich, 1890. These papers may possibly contain more examples.

It will be noticed that these cases occurred in adults, and that the duration varied from nine months to two years. In none of them did metastatic growths occur, the tumours remaining localised, and not growing to any great size. Such cases would presumably be well suited for removal by operation. It would be interesting in future cases to have more information as to the previous occurrence or not of syphilitic infection and of blows or falls upon the back.

To recapitulate the most important features in my case; these were :—

(1) The long duration, with sudden exacerbation of all the symptoms after a fall.

(2) The close resemblance of the sensory disturbance over a certain area to that commonly met with in, and regarded as typical of, syringomyelia. This at the time caused some difficulty in diagnosis.

(3) The remarkable absence of severe pain, and of root-pains, radiating from the affected part of the cord; possibly accounted for by the soft nature and slow progress of the growth producing only very gradual increase of pressure on the nerve roots. This may possibly be in future an important diagnostic point in differentiating this variety of tumour from other growths in the spinal membranes.

(4) The existence after such a considerable time of many apparently healthy fibres in the affected part of the cord and in the degenerated tracts, presumably rendering possible a fair amount of recovery. The operation, however, did not remove the whole of the growth.

A CASE OF CEREBRAL TUMOUR, WITH HEMI-ANÆSTHESIA.

BY H. J. MACKAY, M.D.

James M., aged 45, a labourer, was admitted into Devizes Cottage Hospital on November 20, 1893, complaining of severe headache, inability to walk, and numbness, with some weakness of the left side. His family history was unimportant; his personal record showed that he had chancre in his twentieth year, and malarial fever when on foreign service; otherwise his health had been good, and he had not drunk to excess. His present illness began nine weeks before, with severe pain in the head; he had, however, noticed giddiness and occasional headaches for some months before. Two weeks after the severe headache began he had cramp in his legs, chiefly in the calves, but they did not jerk, merely becoming stiff and painful. During an attack of this sort, seven weeks before admission, he fell downstairs, striking his right ear and the head behind the ear in the fall. A month later he had a shivering attack with intense aching in the back, neck and head (? influenza), for which he went to bed, and remained there till his admission to the hospital. There were no convulsions or motor spasms at any time during his illness, nor did he ever lose consciousness. While in bed he noticed a gradual onset of numbness in the left side of his body, which also became somewhat weaker than the right; coincident with this his wife had noticed increasing torpor and stupidity. He had had severe attacks of vomiting, independently of taking food. He complained also of giddiness, and of failure of sight.

Examination showed the patient to be a well-built, muscular man, who lay on his back with closed eyes, seemed stupid and heavy, and appeared to have difficulty in understanding and answering the questions put to him. When pressed to answer he would, after a pause, say impatiently, "He did not know," or make a quite irrelevant reply. The information he volunteered, however, apart from questioning, was intelligent, coherent, and to the point. He never mixed up incongruous words in his sentences, nor made meaningless sounds. He could read the letters in Snellen's types, and write small words correctly. His condition therefore suggested that of sensory aphasia, of the variety described as word-deafness. He complained greatly of pain in the frontal region, and cranial percussion elicited tenderness over the right temporo-parietal region. His motor power was good on the right side but slightly impaired on the left. He had still, however, considerable power in the left hand grasp, and in flexing and extending the left leg. Muscular nutrition was everywhere good. There was no rigidity on passive movement. Electrical irritability was diminished to both currents on the left side of the body, but there was no qualitative change.

The knee-jerks were normal on both sides, and there was no clonus. The plantar and cremasteric reflexes were impaired on the left side. Bladder and bowel control were normal. The right pupil was dilated to about No. 12 catheter gauge, and did not react to light; the left was of medium size, and re-acted but sluggishly. Co-ordination was unaffected on the right side, but appeared defective on the left.

Muscular sense is much impaired on the left side. He is quite unaware in what position his left arm and leg are placed, imagining them to be straight when they are flexed: this is not the case with the right side.

There is great impairment of sensation to touch, pain and temperature over the left side of the body. He does not feel a pin-prick, which on the right side makes him wince and withdraw the limb. He does not recognise as warm a test tube of water a little below boiling point. He is quite unconscious of light touches over the impaired area. This includes the left half of the face and trunk and the left arm and leg, and the anæsthesia appears to be most marked in the leg. Normal sensation is recovered at about an inch to the right of the mesial line, and continues over the right side of the body.

The condition of the cranial nerves is as follows:—

I. He cannot distinguish in either nostril, tested separately, the smell of hyacinth, cloves, peppermint or tobacco. He complains that there is no flavour in his meat or his tea, and that everything tastes alike.

II. Vision, R. $\frac{6}{60}$. J.o. at 22 cm.
L. P.L. only.

There is optic neuritis on the right side. On the left the disc cannot be examined on account of cloudiness of the cornea. There is concentric contraction of the right field. The right pupil is dilated and motionless, the left is of medium size and probably reacts sluggishly to light. Reaction to accommodation cannot be made out in either eye.

III.
IV.
VI. } Ptosis and paralysis of internal rectus on right side.

V. Nearly complete facial anæsthesia of left side, which includes the mucous surfaces and extends slightly to the right of the middle line. Commencing neuroparalytic ophthalmia in left eye. Masseteric contraction equal on both sides.

VII. Unaffected.

VIII. He hears a watch tick which is normally audible at thirty inches, at a distance of two inches in the right and twelve inches in the left ear. The relatives cannot say whether he was deaf before his illnes, but think he was not. The tympana are healthy in both ears.

IX. He cannot distinguish on either side of the tongue solutions of salt, tartaric acid, quinine or sugar. The tongue is thickly furred. The solutions are applied to the back as well as the front of the tongue.

X.
XI.
XII. } Unaffected.

There is some swelling of the left side of the face, and a bright flush on the left cheek.

T. 99°. P. 64, full, laboured, regular. R. 18.

The urine is acid, specific gravity 1,018, free from albumen and sugar.

On the 23rd it was noted that the ptosis was less marked, and on the 24th the right eye was opened equally with the left, and the right internal rectus had regained its power. The right pupil was still dilated, but now reacted sluggishly to light.

During his stay in hospital the observations made were frequently verified. He was frequently seen to move all his limbs, and to raise himself and sit on the side of the bed. He was unable to stand or walk, on account of vertigo. Subsequently the ophthalmia of the left eye went on to perforation of the cornea, and his torpor deepened; he had still, however, occasional intervals of clearness, when he would ask questions quite intelligently, but could hardly ever be got to return a relevant answer. On December 12 he became comatose, his temperature rose to 106·2°, and he died shortly afterwards.

Autopsy fourteen hours after death, at which I was assisted by Dr. Adie. On opening the skull there was thickening and opacity of the dura over the right temporo-sphenoidal lobe. The arachnoid space contained several ounces of blood-stained serum, which was seen to ooze from the sulcus surrounding a tumour in the right lower temporo-sphenoidal region, and the freedom of the flow suggested communication with the right lateral ventricle.

The tumour (fig. 6) was lenticular in shape, measuring 4·5 cm. in diameter each way, by 2 cm. in thickness. It occupied a bed of softened brain tissue which had absorbed half the breadth of the middle, and had completely obliterated the lower temporo-sphenoidal convolutions. The superior T. S. convolution was greatly enlarged in breadth and length, and was, like the remainder of the brain, displaced upwards and forwards. Owing to this displacement the frontal and parietal lobes were compressed somewhat, and the fissure of Sylvius was anterior to its usual position. Moreover, the T. S. lobe occupied more than its normal proportion of the surface-area of the hemisphere. The left hemisphere was natural in shape and proportion. The nerves at the base of the brain showed no change beyond compression and flattening of the right oculo-motor. No naked-eye change could be detected in the fifth of the left side, nor in the Gasserian ganglion, nor in the auditory of the right side. Unfortunately these structures were not preserved for microscopical examination, as was intended.

On the median aspect of the right hemisphere there is evident compression and softening of the hippocampal gyrus in the neighbourhood of the isthmus, and between this and the uncus. The uncinate, and occipito-temporal gyrus (beneath the collateral fissure), are also superficially softened. The lingual gyrus appears, outwardly, unaffected. The rest of the median surface of the hemisphere appears normal. A vertical (frontal) section was made through the hemisphere in the plane of the centre of the tumour (line A B in fig. 6). This plane passed through the splenium of the corpus callosum in its greatest diameter, and met, at the superior margin of the hemisphere, the posterior and superior extremity of the fissure of Rolando.

It was seen by the section (fig. 7) that the occipito-temporal gyrus was compressed to a mere shred, from 2 to 3 mm. in its greatest thickness, by the tumour. The medullary matter of the lingual gyrus, and that at the bottom of the calcarine fissure (which includes the medullary matter of the hippocampal gyrus at this spot), was softened to pulp. The bed of the tumour occupies most of the thickness of the temporo-sphenoidal lobe in its lower two-thirds and its posterior half; and from the softened medullary matter composing it a strand of softened tissue passes upwards and inwards towards the floor of the lateral ventricle. This softened tract extends up both walls of the lateral ventricle; on the outer side as far as the grey matter forming the bottom of the parallel fissure; and on the inner side, formed here by the cornu ammonis below, and higher up by the alveus of the hippocampus (posterior pillar of the fornix), the softening extends as far as the splenium of the corpus callosum, which is not softened. A small fragment of the posterior extremity of the optic thalamus, which the section includes, lying subjacent to the splenium, is also unsoftened. The cavity of the ventricle is obliterated, and the softened walls are in contact with each other.

A horizontal transverse section through the anterior portion of the hemisphere, in the plane of the line E F (fig. 9), displayed the internal capsule in relation to the softened medullary matter of the T. S. lobe. It was found that the posterior fibres of the capsule did not share in the softening, but were separated from the disorganised tissue by a space, a few mm. in width, of apparently healthy medullary matter. Beyond this distance (about five mm. from the posterior extremity of the lenticular nucleus), the medullary fibres were softened and were in anatomical continuity with the softened medullary substance forming the bed of the tumour. A softened tract of arcuate fibres could also be traced passing into the white matter of the adjacent (superior) T. S. convolution.

Sections in, and anterior to, the plane of C D did not reveal any fresh facts, and sections in the plane of A B through the hinder portion of the brain showed that the area of softening did not extend posteriorly beyond the region of actual compression by the tumour.

The fibres of the crusta and tegmentum of the cerebral peduncles showed no visible change, nor did the longitudinal fibres of the pons.

The brain was hardened in Müller's fluid for two months, and for a month in alcohol. Sections were then cut from: (1) the tumour; (2) the posterior fasciculi of the internal capsule; (3) the tract of medullary substance posterior to this, endeavouring to include the boundary line between healthy and softened tissue; (4) the softened hippocampus; (5) the superior T. S. convolution in its middle undamaged portion; (6) the right crus-cerebri and pons.

(1) The tumour was found to be enclosed, on its outer

surface, in a capsule of vascular connective tissue, which appeared to be thickened pia. Internal to this was a zone containing numerous arterioles surrounded by connective tissue which was densely infiltrated by small-celled leucocyte-looking bodies which took on a deep stain, and resembled the granulation tissue of syphilitic growths. The arterioles in this zone and in the capsule were for the most part either obliterated or in process of being obliterated, by the invasion of the outer and inner tunics by the small-celled growth mentioned. Many of those so obliterated showed the presence of white fibrous tissue more or less replacing the walls of the vessel, and some of them on section had the appearance of being round cords of fibrous tissue. More internal to this granulation zone were areas of loose connective tissue, and strands of dense fibrous tissue, the latter sometimes enclosing tracts of hæmorrhagic *débris*, and elsewhere of caseation.

(2) The posterior fibres of the internal capsule showed no morbid change. The nerve fibres were of equal size; their axis cylinders took on a deep stain with aniline blue black. The unstained white sheaths exhibited no enlargements nor degeneration.

(3) The tract of softened tissue posterior to the internal capsule could not be included in sections even when cut in celloidin. The broken edges from which tissue had disappeared exhibited at the boundary line swollen and irregular fibres, and numerous dark stained connective tissue nuclei. Sections teased in osmic acid showed abundant myelin globules in the irregular fibres, compound granule bodies and fatty *débris*.

(4) The hippocampal sections showed a total disappearance of the longitudinal fibres forming the alveus, and an absence of the large pyramidal cells from the subjacent layer. There appeared to be a great increase of connective tissue, with darkly stained nuclei, but the normal arrangement of the other layers in this part of the brain could not be recognised.

(5) The superior temporo-sphenoidal convolution was not softened anywhere except in the medullary (arcuate)

CASE OF J. M.

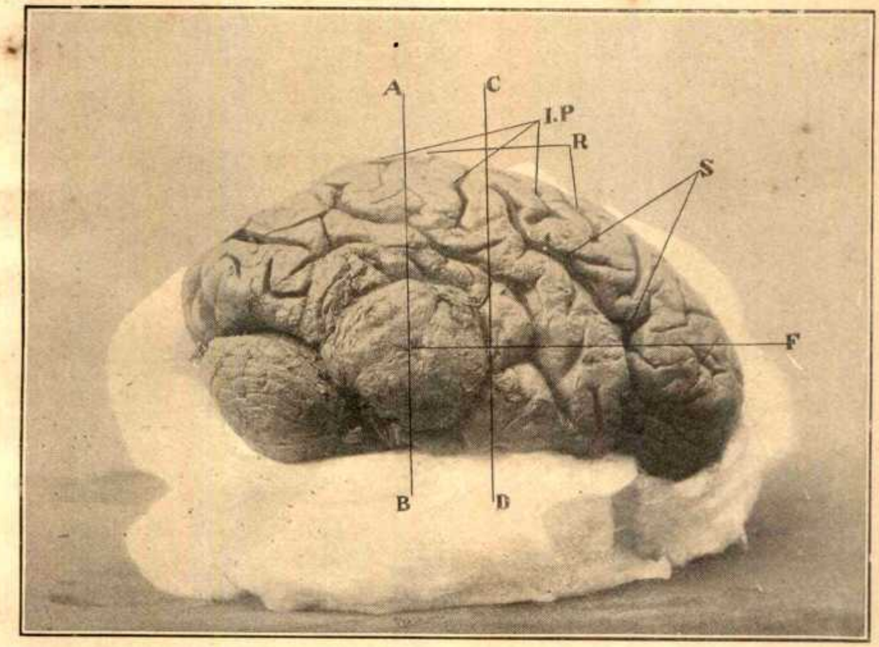


FIG. 6.

Right Hemisphere, with Tumour *in situ*. From photograph.

To show the tumour more clearly, the point of view is from a little below the transverse axis of the brain. Consequently the upper margin, including the termination of the fissure of Rolando, is not visible. I.P points to inter-parietal fissure; R and S, fissures of Rolando, and of Sylvius; the lines refer to planes of section mentioned in the text.

CASE OF J. M.

Section through hemisphere in plane of line AB. From a photograph, nat. size.

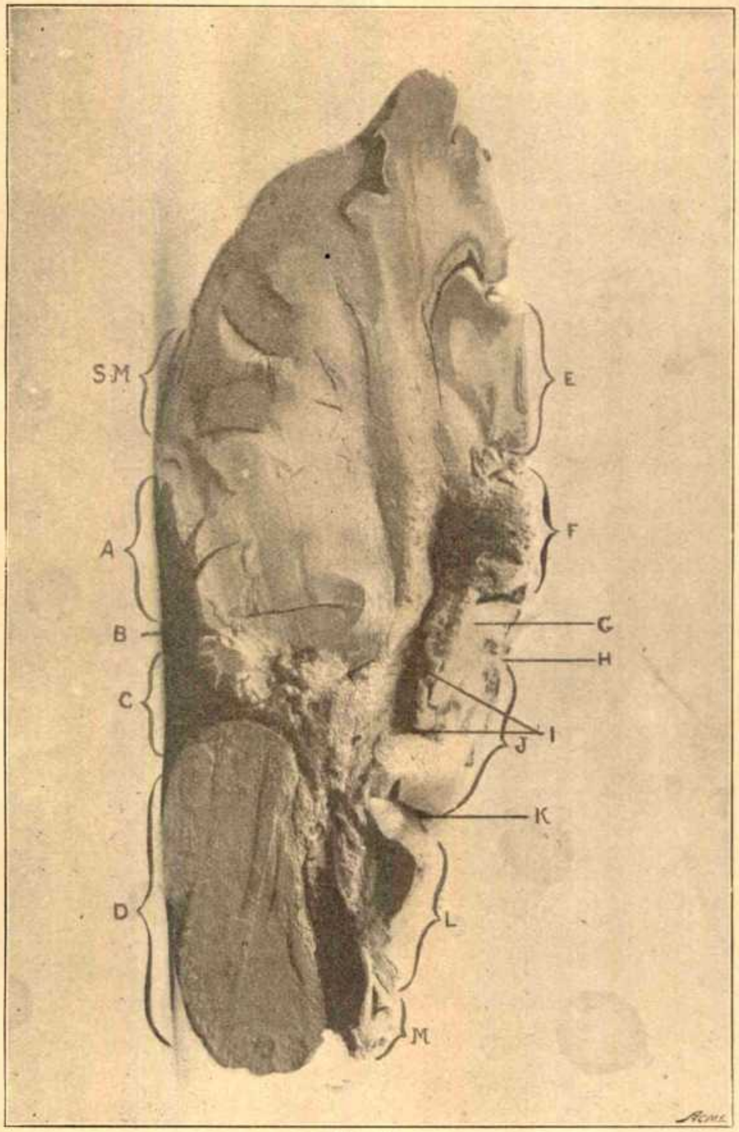


FIG. 7.

(The letters refer to figs. 7 and 8.)

SM—Supra-marginal Convolution; A—Angular Convolution; B—Parallel Fissure; C—Middle Temporo-sphenoidal Convolution; C^1 (in fig. 8)—Inferior T.S. Convolution; D (in fig. 7)—Tumour; M—Occipito-temporal Gyrus; L—Lingual Gyrus; K—Calcarine Fissure; J—Hippocampal Gyrus; I—Hippocampus Major; H—Hippocampal Fissure; G (in fig. 8)—Posterior Extremity of Optic Thalamus.

Healthy Brain.

Section in same plane as in fig. 7, for comparison. From a photograph, nat. size.

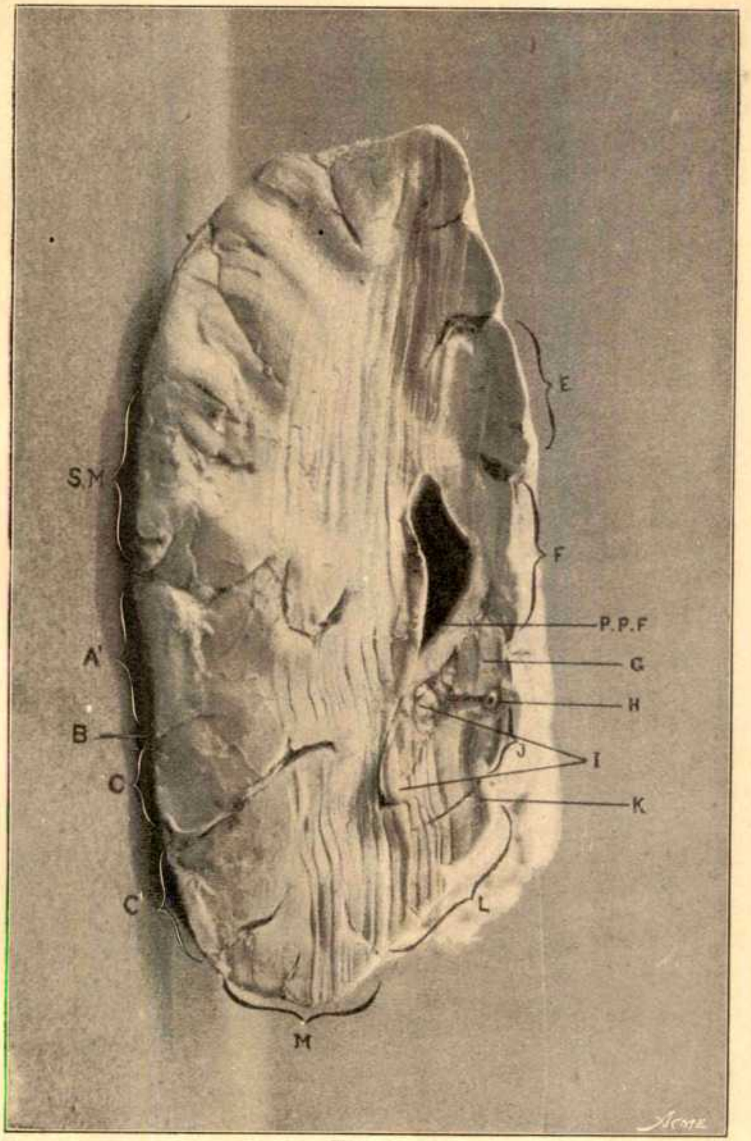


Fig. 8.

(The letters refer to figs. 7 and 8.)

P.P.F—Posterior Pillar of Fornix, descending to become the Alveus of the Hippocampus. The same structure, but disorganised, can be traced in fig. 7; F—Splenium of Corpus Callosum. The softened appearance in fig. 7 is due to compression by cotton wool, which was packed into the upper part of the Ventricle; E—Callosal Convolution.

CASE OF J. M.

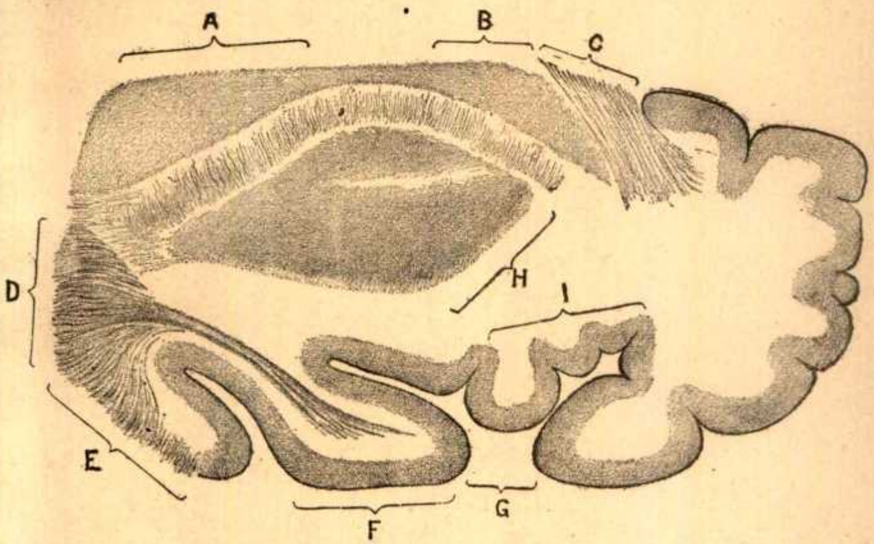


FIG. 9.

Horizontal transverse section through anterior portion of Hemisphere in plane of line E F. (From a tracing.) The softened medullary tissue at D is continuous with E, the softened bed of the tumour. There is seen to be an interval of unsoftened medullary substance posterior to the internal capsule. H—Lenticular Nucleus; B—Caudate Nucleus; A—Optic Thalamus; C—Commissural fibres of genu of corpus callosum; F—1st T.S. Convolution, into which passes a strand of softened fibres; G—Fissure of Sylvius; I—Insula.

fibres in its anterior portion already referred to. Elsewhere there was a great increase in the size, and in the relative mass, of the neuroglial fibres and cells. The nuclei were more numerous than in normal sections and were occasionally double.

(6) The crus cerebri and pons exhibited normal fibres, with unaltered sheaths and axis cylinders on transverse section.

The sections were stained for the most part by logwood and carmine, by Bevan Lewis's method, and by osmic acid. I regret that my want of skill as a microscopic draughtsman does not allow me to reproduce their appearance.

Remarks.—The dearth of clinico-pathological records, throwing any light upon the localisation of the cerebral sensory areas, has been frequently remarked. The reproach cannot even yet be said to be in a fair way of being removed—a search of the only available literature at my disposal having yielded but scanty notes of cerebral cases in which tactile anæsthesia was associated with cortical lesions.

Ferrier[1] reports a case in which a tumour pressing on the left temporal and parietal lobes caused right hemianæsthesia. Vialet[2] mentions two cases of left hemianopsia in one of which right hemi-anæsthesia was present. In both of these there was atrophy of the convolutions bounding the calcarine fissure, atrophy of "the central portion of the parieto-occipital fissure," and atrophy of the foot of the cuneus extending to the lower extremity of the hippocampus; unfortunately no detailed criticism is given of the association of symptoms with lesions in each individual case.

Dana,[3] in a collection of one hundred and ninety-two cases. quotes twenty as being cases in which no anæsthesia was associated with lesions of the gyrus fornicatus or gyrus hippocampi. By his own showing, however, some disturbance of sensation was present in nearly one-third of these cases.

Savill[4] reports left anæsthesia and trophic changes occurring in a case in which a cavity was present beneath the cortex of the right gyrus fornicatus.

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[1] *Lancet*, ii., 1887.

[2] *Medical Week*, vol. i., No. 34.

[3] *Journal of Mental and Nervous Diseases*, Oct., 1888.

[4] BRAIN, 1890.

Hammond[1] reports a case in which (among other lesions) pressure on the left gyrus fornicatus was associated with anæsthesia of the same side.

If clinical observation, however, lags far in rear of experimental research in this matter, it is satisfactory to note that the direction of the former is in the footsteps of the latter. Thus Horsley and Schäfer,[2] expanding Ferrier's previous conclusions, are of opinion that the gyrus fornicatus represents, in the hemisphere, the area of cutaneous sensibility, and on the clinical side, Mills, after an exhaustive digest of recorded cases,[3] concludes that "no part of the brain is more likely to contain the areas (for cutaneous sensibility, *i.e.*,) than the gyrus fornicatus, the hippocampal gyrus, the precuneus, and the postero-parietal convolutions.

It should be encouraging, too, to remember, that the difficulties which beset a solution of the problem do not differ, in kind or in degree, from those which have hindered the settlement of the question of motor localisation. They arise chiefly from cases, apparently anomalous, in which the specific symptoms are associated with lesions of areas other than those indicated by experiment, and in the second place, from instances, apparently negative in import, in which morbid conditions of the regions indicated by experiment are accompanied by no specific symptoms. Concerning the latter, the phenomena of functional compensation are probably equally valid as explanatory of the apparent anomalies of sensory "negative instances" as they have been shown to be in the case of motor functions. And as regards the first-mentioned difficulty, the accumulation of recorded cases will probably, with a widening knowledge, reveal the explanation of these present-seeming discrepancies.

In the case I have now recorded, the ascertained absence of morbid change from the sensory tracts of the pons, crus, and internal capsule, points to the cortical areas involved, and the parts underlying them as the seat of the sensory disturbance.

[1] *Ib*, 1891. [2] *Trans. Roy. Soc.*, 1888. [3] BRAIN, 1889.

The location of the softened tract posterior to the internal capsule is such that it would involve the temporal portion of the temporo-occipital cerebellar fibres, which, lying to the outside of the pyramidal fibres in the crusta, are known to radiate outwards and backwards to the temporal and occipital cortex. As these fibres are presumed to be motor, or at least not sensory in function, however,[1] the sensory impairment cannot be associated with their damage. It is reasonable, therefore, to connect the anæsthesia, general and special, with the cortical lesions on the outer and mesial surfaces of the temporal lobe, and with the destruction of the subjacent medullary tissue. Such a conclusion would be in complete accord with the observations of Ferrier and Yeo,[2] who showed that lesions of the hippocampal gyrus, hippocampus, and the neighbourhood of the calcarine fissure, were associated with loss of sensation on the opposite side of the body, and, inasmuch as the hippocampal and uncinate gyri are merely subordinate divisions of the gyrus fornicatus, the conclusion is equally in harmony with the later observations of Horsley and Schafer.[3]

With reference to the special senses, it is probable that the loss of hearing on the right side was due to intra-cranial pressure on the soft fibres of the right auditory. The right superior T.S. convolution was not affected in its posterior part, otherwise than by being displaced upwards, and by an apparent hypertrophy of connective tissue.

The impairment of taste and smell on the left side were probably contributed to, if not accounted for, by the facial anæsthesia on that side. On the right side, however, the loss may be associated with the damage to the grey and medullary matter of the hippocampal gyrus consequent on the pressure of the tumour.

In considering the almost total amblyopia of the left eye, the difficulty is to assign to each of several morbid conditions present, its legitimate share in the result. There was extreme cloudiness, with, later, ulceration of the cornea.

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[1] Gowers, "Nervous Diseases," vol. ii, pp. 29 and 65.

[2] "Functions of Brain," 1886, chap. ix.

[3] *Supra cit.*

It is possible that optic neuritis was also present. I also refer, in this connection, but with diffidence, to possible destruction of fibres of the optic radiation of Gratiolet in the white matter of the temporal lobe, in the area of softening described. I am not aware whether this lesion has ever been associated definitely with crossed amblyopia.

The association of the latter condition with lesions of one angular gyrus has been established by Ferrier,[1] and clinical cases of similar import have been recorded by him, by Grasset,[2] and by Sharkey.[2]

A case of hemianæsthesia, with homonymous hemiopia; in which the softening involved the fibres of the optic radiation, is reported by Beevor;[3] but in his case the area of softening was much more extensive than in mine, extending back to the surface of the occipital lobe.

If the fibres of the optic radiation proceed, in part, to the angular gyrus, as well as to the occipital visual centres, it seems possible that a lesion of small area might affect those fibres only, or chiefly, which are in connection with the angular gyrus of the same side, and so produce crossed amblyopia rather than hemianopia. Although, in my case, the patient's condition rendered futile any attempt at a careful perimetric investigation, yet I am strongly of opinion from the observations made that his visual condition indicated crossed amblyopia rather than hemiopia.

The condition of vision in the right eye was sufficiently accounted for by ascertained optic neuritis.

The patient's mental condition which, on his admission, I thought resembled the condition described as word-deafness, is extremely interesting in connection with Starr's investigations into the pathology of sensory aphasia.[4] In an analysis of fifty cases of sensory aphasia, Starr found that the convolutions most frequently affected were the first and second temporo-sphenoidal. In seven cases pure word-deafness was present, and in all of these the lesion was limited to the first and second T.S. convolutions, in their posterior two-thirds.

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[1] *Op. cit.*, p. 287.

[2] *Op. cit.* 290. I have not had an opportunity, however, of referring to the originals.

[3] *Lancet*, May 5, 1895.

[4] BRAIN, 1889.

As regards diagnosis, I may say that the only approach to one made during life was the suspicion of a gummatous tumour or tumours, somewhere at the base of the brain. The difficulty, of course, was to harmonise signs which pointed at once to compression of the right oculo-motor, and auditory, with injury to the left trigeminal; and to explain at the same time interference with the sensory tract passing to the left side of the body, without serious implication of the motor fibres. Due weight was perhaps not given to the olfactory indications which might have led to a localising diagnosis of injury in the region of the uncinate and hippocampal lobules, nor were there present to my mind the localising indications afforded by sensory aphasia, according to Starr, and quoted above. Needless to say, no single lesion could be imagined associating all the signs present, and the invocation of multiple lesions did not afford much assistance. It is to be hoped that sufficient clinical material will eventually be accumulated to allow of greater confidence in the localisation of the cerebral sensory areas, and the present case is recorded with a view to that end.

ON DIPHTHERITIC PARALYSIS.

Being an Analysis of the Cases occurring at the Eastern Hospital, Homerton, during the Years 1892 *and* 1893.

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Before entering upon the subject-matter proper of this paper, there are a few preliminary observations which I desire to make. Firstly, with respect to the nature and circumstances of the cases of diphtheria that have been admitted into the hospital during the two years 1892 and 1893: the patients came almost entirely from the eastern and north-eastern districts of London, and were furnished mostly by the lower and lower-middle classes of society (labourers, mechanics, small shopkeepers, clerks, &c.). The severity of the cases, judged by the mortality, was the same each year; the mortality per cent. (calculated according to the Registrar-General's formula) was 35·6 for 1892 and 35·2 for 1893. The cases, both of diphtheria and of paralysis, were consecutive. All cases of disease sent to the hospital certified to be diphtheria, but found to be of another nature, are excluded.

Secondly, with respect to the length of time the patients were detained in the hospital: it has been my practice (and it has been rigidly carried out) not to discharge any child before the end of six weeks from admission, unless the attack of diphtheria began any length of time before admission, which, however, is very exceptional. Adults are detained for six weeks only when the attack is severe or when some complication arises to prevent their discharge; otherwise, they are allowed to leave at the end of four weeks at the earliest. A considerable number of patients have been sent, at the end of three or four weeks, to the Northern (Convalescent) Hospital at Winchmore Hill, where they have remained for a further period of three or four weeks. A few of these patients have been attacked with paralysis while at Winchmore Hill; Dr. Hume, the medical superintendent of the Northern Hospital, has kindly supplied me with information concerning these cases, and they are included in my list.

Thirdly, nearly all the cases of diphtheria, and still more of paralysis, have come under my own personal observation; but for the bulk of the written notes I am much indebted to the assistant medical officers of the hospital, who have taken much trouble over them.

I have made these remarks in order that others may get some idea of the class of case with which this paper deals, and of the conditions under which the cases have been observed. It will be found that some of my statements differ from those made by other writers on the same subject. But, inasmuch as the experience derived from one hospital may differ widely from that of another, and both from that gained in private or consulting practice, it is not surprising to find even marked difference of statement with respect to what are supposed to be questions of fact, let alone of opinion. The truth can only be arrived at by a combination of experiences.

Number of Cases.—During the years 1892 and 1893, 1,071 cases of primary diphtheria[1] have been under treatment in the hospital, including those remaining at the end of 1891 and excluding those remaining at the end of 1893. Amongst these cases there have been 125 instances of paralysis, an incidence of 11·6 per cent. on the total cases; 17 of the 125 cases proved fatal. It has to be remembered, however, that in diphtheria death, due to the attack itself, takes place fairly early—before, to employ Roger's expression, the patient has time to become paralytic. Hence, a fairer estimate of the frequency of occurrence of paralysis will be obtained if the fatal cases (less, of course, those fatal during paralysis) be subtracted from the total, and the incidence be calculated upon the remainder. There were 362 such fatal cases, so that 709 patients survived to run the risk of becoming paralytic; 125 were so affected—an incidence of 17·6 per cent.

Sex.—Of these 125 patients, 55 were males and 70 females—a proportion of 1 male to 1·27 females. This slight preponderance of females may possibly be accounted for as follows:

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[1] As recognised clinically; by primary is meant not secondary to scarlet fever, measles, &c.

more females than males were under treatment—the 1,071 cases being made up of 478 males and 593 females (one male to 1·24 females); moreover, the mortality was higher among males than females—183 of the former and 179 of the latter having died (excluding deaths due to or occurring during paralysis), so that 295 males and 414 females lived to run the risk of paralysis (1 male to 1·4 females); seeing, however, that 55 of these males and 70 of these females did become paralytic, it would seem that males are a little more likely to be so affected than females, for the incidence in the former sex is 18·6 per cent.; in the latter, 16·9.

Age.—The numbers of cases are arranged in the following table according to age:—

Age.	*No. of Cases.*
Between 1 and 2 years	2
,, 2 ,, 3 ,,	7
,, 3 ,, 4 ,,	13
,, 4 ,, 5 ,,	13
,, 5 ,, 6 ,,	17
,, 6 ,, 7 ,,	17
,, 7 ,, 8 ,,	11
,, 8 ,, 9 ,,	8
,, 9 ,, 10 ,,	8
,, 10 ,, 11 ,,	8
,, 11 ,, 12 ,,	6
,, 12 ,, 13 ,,	4
,, 13 ,, 14 ,,	2
,, 14 ,, 15 ,,	3
,, 17 ,, 18 ,,	2
,, 23 ,, 24 ,,	1
,, 26 ,, 27 ,,	1
,, 28 ,, 29 ,,	1
,, 42 ,, 43 ,,	1
	125

Thus the ages of the patients range from one to forty-two years; there were none under one year of age, and only 9 under three and 6 over fifteen years. Most of the patients were, however, under ten years, as will be seen from the following table, in which the cases are arranged in periods of ten years:—

Under 10 years	96
10 to 20 ,,	25
20 ,, 30 ,,	3
30 ,, 40 ,,	0
40 ,, 50 ,,	1
	125

These figures differ widely from those given by Landouzy in 68 cases quoted by Dr. Gowers in his treatise on "Diseases of the Nervous System" (2nd edition, vol. ii., p. 903). The statements Dr. Gowers makes, apparently in consequence of Landouzy's figures, that "adults furnish a larger proportion of the cases of paralysis than of diphtheria," and that "it would seem that the tendency to paralysis is very slight in early infancy and that it increases the older the sufferer," appear to me to require some modification. No doubt the statements are in accordance with Landouzy's figures, but there must have been some factors in operation causing adults rather than children the subjects of diphtheritic paralysis to be brought under Landouzy's notice. If we arrange the 1,071 cases of diphtheria in ten-yearly age groups with the deaths at the corresponding periods we obtain the following table:—

Under 10	years,	780	of whom	359	died,	15	after paralysis
10 to 20	,,	176	,,	12	,,	1	,,
20 to 30	,,	73	,,	4	,,		
30 to 40	,,	30	,,	2	,,		
40 to 50	,,	9	,,	1	,,	1	after paralysis
over 50	,,	3	,,	1	,,		
		1,071		379		17	

Comparing this with a similar table for the cases of paralysis given above we find that of 780 patients under 10 years of age, 96 became paralysed, or 12·3 per cent.; of 176 between 10 and 20 years, 25 became paralysed, or 14·2 per cent.; and of 115 of 20 years of age and upwards, 4 became paralysed, or 3·4 per cent. But the fairest way to get at the incidence of paralysis is (as has been before stated) to subtract from the total cases of diphtheria all those that proved fatal, with the exception of those fatal after the onset of paralysis. If this be done we have 436 cases under 10 years of age, of whom 96 became paralysed, or 22 per cent.; between 10 and 20 years of age there are 165 cases, of whom 25 became paralysed, or 15·1 per cent.; above 20 years of age there are 108 cases, of whom 4 became paralysed, or 3·7 per cent. Again, of 125 cases of diphtheritic paralysis 96 or 76·8 per cent. were children under 10 years of age; 25, or 20 per cent. were aged from 10 to 20 years, and 4, or 3·2 per cent. were more than 20 years old; and of the 1,071 cases of diphtheria, amongst which the cases of paralysis arose, 73 per cent. were under 10; 16 per cent. between 10 and 20, and 11 per cent. were above 20. On the question of age incidence De Gassicourt's treatise on the "Diseases of Childhood," and the work on "Peripheral Neuritis" by the late Dr. Ross and Dr. Bury, should be consulted.

The following table gives these figures concisely :—

Age.	Number of cases of diphtheria.	Deaths, excluding deaths after paralysis	Number of cases *minus* number of deaths.	Number of cases of paralysis.	Percentage of cases of paralysis on cases *minus* deaths.	Percentage of cases of paralysis on total cases.	Number of deaths after paralysis.
Under 10	780	344	436	96	22·0	12·3	15
10 to 20	176	11	165	25	15·1	14·2	1
20 to 30	73	4	108	4	3·7	3·4	0
30 to 40	30	2					0
40 to 50	9	0					1
50 and upwards	3	1					0
Total ..	1071	362	709	125	17·6	11·6	17

Date of onset of Paralytic Symptoms.—In 101 of the cases the date of commencement of the attack of diphtheria is definitely stated as well as the date of the first appearance of paralytic symptoms. I find that the seventh is the earliest day upon which such symptoms have been observed, and the forty-ninth is the latest; taking the weeks in order, in 2 cases paralysis set in during the first week, in both cases on the seventh day, in 22 cases during the second week, in 28 during the third week, in 22 during the fourth week, in 15 during the fifth week, in 8 during the sixth week, and in 4 during the seventh week.

Usually the membrane or exudation clears off completely before the paralytic symptoms set in; but not always. I have notes of 84 cases in which the length of time during which exudation remained upon the fauces or in the nasal passages was recorded; in 10 of these cases the exudation had not disappeared when the paralysis commenced (for instances, see Cases 5 and 21).

Muscles first affected.—With respect to the parts in which the symptoms of paralysis first show themselves, in 83 cases the palate was first affected, in 20 the ciliary muscles, in 5 the palate and ciliary muscles, in 4 the palate and lower extremities, in 3 the lower extremities, in 2 the muscles of the pharynx, and in 2 the respiratory muscles. In the remaining 6 cases the parts first affected were as follows, there being one instance of each: palate and upper extremities; ciliary muscles and lower extremities; pharynx and lower extremities; lower extremities and ocular muscles; muscles of trunk and neck; muscles of trunk and extremities. It will thus be seen that in a large proportion of the cases (66·4 per cent.) the palate alone is the first part to be affected; while either alone or in combination with some other muscles it is the first part to suffer in 74·4 per cent. of the cases.

Extent of Paralysis.—In 28 cases the paralysis remained limited to the palate; in 17 to the ciliary muscles[1]; in 11 to the palate and ciliary muscles; in 6 to the palate and lower extremities, including one case in which the vagus was probably also involved (Case 14); in 3 to the palate and one or more ocular muscles; and in 1 to the respiratory muscles including the larynx; in this case too the upper extremities were slightly affected (see Case 10). Whether the vagus or its branches ever become affected alone is a question I shall discuss later. From the above list of cases it will be seen that in 66 of the 125 cases the paralysis was limited in extent, that is, in 52·8 per cent. It is very likely that a few cases of ciliary paralysis have been undetected, many of the patients being young children. All who are old enough to know their letters are tested regularly with Jager's types. Myopia also conceals slight ciliary paralysis.

In 16 cases the paralysis was generalised.

Frequency with which certain Muscles or Groups of Muscles are affected.—In 102 cases, the palate was paralysed; in 56, the ciliary muscles; in 52, the lower extremities; in 26, one or more of the external ocular muscles; in 21, the upper extremities; and in at least 11, the muscles of the larynx.

In 19 cases, difficulty in swallowing was a marked symptom; but this number does not by any means represent all the cases in which the pharyngeal muscles were paralysed. In all cases where there is marked regurgitation of fluids through the nose, the œsophageal tube is employed for feeding; hence subsequent pharyngeal paralysis is undetected.

Of the 26 cases in which the *external ocular muscles* were affected, in 14 the notes only state the presence of squint, without specifying the exact muscle or muscles paralysed. In 7 of the remaining 12 cases, one external rectus (usually the left) was paralysed; in 3, both external recti; and in 2, most of the muscles of the eyeballs (Case 12 was one of these). In none of the cases were the internal recti affected apart from the external. In Case 12 there was left ptosis.

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[1] No attempt has been made in any of the cases to determine whether only one or both ciliary muscles were affected.

In 10 cases there was evidence of paralysis, more or less complete, of the *diaphragm;* 4 of these were fatal cases.

In 11 cases there was what I have taken to be evidence of paralysis of the *adductors of the vocal cords*, viz.: aphonia, with a toneless and ineffectual cough. Five of these cases were fatal. In 6, the diaphragm was also affected.

In 3 cases the *abductors of the larynx* appeared to be paralysed. The following are the notes of the most marked example:—

Case 1.—Annie G., aged 3½ years, admitted on May 28, having been ill since the day before. There was a little deposit on the right tonsil, and slight laryngeal obstruction. Seven hours after admission tracheotomy was performed, the laryngeal obstruction having become much worse. Membrane was removed from the trachea, and the relief afforded was not immediately complete; but the next day the patient was very comfortable. On May 31, a good deal of membrane was coughed up through the tube. On June 1, the tonsil was clear of deposit.

There was albuminuria on June 3, and this continued till August 7.

On June 16, it was noted that the child could not drink, as violent coughing was set up. Since the operation she had been fed with a tube passed into the stomach. The tracheotomy wound was quite healed by June 23, and by June 27 the child was able to take nourishment in the usual way without any difficulty. On July 2, the child was unable to sit up in bed, and there was a slight squint.

July 3.—"Some cough during the morning. At 11 a.m., sudden cyanosis and dyspnœa. Much recession of supra-clavicular spaces, but not much of chest wall." This attack of dyspnœa was so urgent that the assistant medical officer, Mr. H. E. Mann, who was called to the patient, had to perform tracheotomy, and insert a tube without delay. Complete relief was afforded by this measure. There was no membrane in the trachea.

July 7.—"The patient is quite unable to breathe through the larynx."

July 10.—"The tube was omitted yesterday. To-day, the child is breathing through the mouth without difficulty." By July 14, the wound was healed. On August 1, it was noted that the paralysis was improving; the patient could stand without assistance; drink did not return through the nose; the voice was still nasal, though not so much as before.

August 8.—"The patient is improving, but there is still some stridulous breathing during sleep." She remained in about the same condition till August 26, when the following note was made:—"Patient had an attack of urgent dyspnœa early this morning, which appeared to be spasmodic, and during which she became very blue. On the acute paroxysm passing off, considerable stridor remained, and some recession of the chest walls." Further dyspnœa set in later. Intubation of the larynx was then performed, and relief afforded. The child passed a comfortable night. The tube was removed twenty-four hours after insertion; the child could breathe easily. She never had any further respiratory trouble, except on the night of September 2, when the breathing was observed to be harsh. She left the hospital well on October 8.

No note has been made about the character of the voice, but I distinctly remember that it was gruff before the second tracheotomy, and during the period between the second tracheotomy and the intubation, and for some time after. There was no evidence that the secondary respiratory difficulties were due to any reformation of membrane. There was no vomiting or cardiac irregularity.

In none of the cases was *facial paralysis*, paralysis of the *tongue*, or of the *sphincters of the bladder* or *rectum* observed.

Cardiac Irregularity.—In 29 of the cases it was noted that at some time or other during the paralysis there was irregularity of the cardiac rhythm, usually with increased frequency of beat. This condition was met with chiefly in the severer cases of paralysis, but sometimes it occurred in connection with very slight and limited palsy, such as of the palate or ciliary muscles. Indeed, cardiac irregularity occurs after diphtheria quite apart from paralysis. The duration of the cardiac disturbance may be from a few days to several weeks. Sometimes there is dilatation of the heart.

Respiratory Irregularity.—Very occasionally the respiratory rhythm is observed to be altered. It was so in four cases, of which two were fatal—Cases 10 and 11, and perhaps, too, Cases 6 and 14 (see also Case 24). The following are the notes of the two cases which recovered:—

Case 2.—Lillie M., aged 3 years, sister of Case 8, admitted July 10, having been ill since the 8th. There was much membrane on the palate and tonsils, which did not clear off for several days. There was a faint trace of albumen in the urine. The amount of albumen increased so that by July 22 the urine was loaded. On this date the quantity of urine passed in the twenty-four hours was only one ounce, and the quantity continued to be scanty for the following four or five weeks. The urine was not free from albumen till October 18. The first symptom of paralysis was a nasal voice on July 26. Gradually there supervened cardiac irregularity, and paralysis of the upper and lower extremities, of the trunk, of the left external rectus oculi, and of the ciliary muscles. On August 27, she had attacks of irregular breathing, the respirations occurring in sighs and gasps. The diaphragm appeared to act very imperfectly. The attacks of irregular breathing were followed by periods of quiet, regular breathing, in which the respiratory rate was 32 per minute, and the pulse 128. After this, the patient gradually improved, and was well by the end of September.

Case 3.—Sidney S., aged 2½ years, admitted March 11, having been ill since the 6th. There was membrane on the soft palate, which persisted for a week after admission; there was also a discharge from the nose and slight laryngeal symptoms, which were present till March 23. There was albumen on one occasion only—March 17. Paralysis set in on March 24, with deficient movement of the thorax; next day it was observed that he had symptoms such as I have above ascribed to paralysis of the adductors of the vocal cords. Pulse, 142; respirations, 60. On March 26, the breathing was irregular, respiration consisting of a number of rapid, followed by a series of slower, respiratory acts. The average rate was 40 per minute; pulse, 140. On the 28th, the breathing was still irregular, but less so than before. Subsequently, the trunk and neck muscles became partially paralysed, and also those of the upper extremities, and, to a slight extent, those of the lower. The intercostal muscles and diaphragm also became affected, and still acted imperfectly as late as April 28. By the beginning of June paralytic symptoms had quite disappeared.

It will be observed that in both these cases as well as in the fatal cases (numbers 10 and 11) and Case 24 related hereafter, irregularity of respiratory rhythm accompanied paralysis of the respiratory muscles, slight in Case 24.

Sensory Disturbances are, perhaps, more common than is supposed. Those most frequently met with are sensations of "pins and needles" or of tingling in the fingers and toes, and numbness of the same parts. In such cases careful testing will elicit the presence of partial loss of ordinary sensation. In the case of children, however, it is very difficult to be certain about slight sensory changes. As most of my patients were children, I have notes of very few cases of sensory disturbance. The following is an account of the best example :—

Case 4.—Alfred H., aged 28 years, admitted on July 11, having had a sore throat since July 9. There was much swelling of the tonsils and palate; the tonsils were covered with thin membrane. The breath was fœtid. During the next two or three days the membrane spread to the palate and uvula, and there was a discharge from the nose. The fauces were not clear till July 22. There was a good deal of albumen in the urine from the day of admission till the end of July. The urine was not free from albumen till August 24.

The first symptom of paralysis was inability to read small print on August 2. On that date the knee-jerks were brisk. The next was a feeling of numbness in the tongue, first noticed on August 15; this lasted for about a week. Towards the end of the third week in August other sensory and motor disturbances appeared which progressed during the next fortnight. On September 8, the following note was made:—"During the past fortnight the patient has complained of weakness and numbness of the left arm. To-day there is a marked deficiency in the power of the muscles of the left forearm and hand; also slight deficiency in the muscles of the upper arm. The power of the left little finger is very weak, that of the ring finger less so, and still less that of the remaining fingers and the thumb. But the muscles of all these digits are decidedly weak when compared with those on the right side. There is loss of common sensation on the palmar aspect of the left little and ring fingers, and this condition extends up on to the corresponding part of the palm and forearm. On the dorsal aspect sensation is only impaired. Both knee-jerks are present. There is no weakness of the lower extremities. The elbow and wrist-jerks on each side are present but feeble. No anæsthesia of right upper or of lower extremities. No motor paralysis of right arm. He can read J. 4. The patient has in fact slight ciliary paralysis, and partial left brachial monoplegia."

During the next few days the ciliary paralysis disappeared. But the right upper extremity became partially paralysed and the man complained of numbness of the feet. On September 20, it was noticed that the knee-jerks were absent, and his gait was ataxic. He complained of pain in the legs below the knees, especially at night. There was still numbness of the feet. The grasp of the right hand was very weak, that of the left *nil*. The patient then gradually improved, and left the hospital well on October 23.

Fatal Cases.—An analysis of the 17 fatal cases shows that in 13 the cause of death was to be put down to paralysis. Six of these cases proved fatal from cardiac failure; 4 from respiratory paralysis; 2 from persistent vomiting and cardiac failure, and 1 from convulsions. In these 13 cases death took place at times varying from twenty-nine to sixty-two days after the onset of the attack of diphtheria, and on the third to the thirty-fourth day of the paralysis.

In 2 of the remaining 4 cases death was due to the severity of the attack of diphtheria, and took place on the fifteenth and nineteenth day of the disease, and the fourth and sixth of the paralysis, which was limited to the palate in each case. In the third case death was due to a relapse of diphtheria occurring when the patient was recovering from paralysis; while in the fourth case, Case 21, death did not supervene till the thirty-eighth day of the disease, yet membrane persisted upon the fauces, and death was probably due to the direct effects of the poison of diphtheria. Fifteen of the 17 deaths were in patients under 10 years of age.

Albuminuria.—In 122 out of the 125 cases a record has been made as to the presence or absence of albumen in the urine. In only 12 cases has albumen been absent. In 2 out of the 110 cases in which albumen was present, no note has been made as to the length of time during which the albuminuria lasted. In 20 of the remaining 108 cases the duration of the albuminuria was less than one week; in 88 it was for a period varying from one to thirteen weeks. The amount of albumen present was very variable, both in different cases, and in the same case at different times.

Duration of Paralysis (excluding the fatal cases).—In 96 cases it was noted with tolerable accuracy how long the attack of paralysis lasted. In the following table the results of the analysis of the notes in this respect are shown:—

Under one week	6 cases
Between 1 and 2 weeks	1 case
,, 2 ,, 3 ,, ...	3 cases
,, 3 ,, 4 ,, ...	5 ,,
,, 4 ,, 5 ,, ...	7 ,,
,, 5 ,, 6 ,, ...	9 ,,
,, 6 ,, 7 ,, ...	15 ,,
,, 7 ,, 8 ,, ...	21 ,,
,, 8 ,, 9 ,, ...	7 ,,
,, 9 ,, 10 ,, ...	5 ,,
,, 10 ,, 11 ,, ...	8 ,,
,, 11 ,, 12 ,, ...	4 ,,
,, 12 ,, 13 ,, ...	2 ,,
,, 13 ,, 14 ,, ...	2 ,,
,, 14 ,, 15 ,, ...	1 case

In none of the cases was any permanent paralysis left behind. There is no intermediate result between death and recovery.

With respect to the *connection between the amount of exudation upon the fauces and the subsequent paralysis*, the cases have been divided into four groups :—

(1) Those in which there was no exudation—3 in number.
(2) Those in which the amount was slight—24.
(3) Those in which there was a moderate amount—45.
(4) Those in which there was much exudation—53.

These observations, of course, only apply to the condition of the fauces as seen after admission to the hospital. It is quite possible, for instance, that in the 3 cases in which no exudation was observed on or after admission, that it had been present before admission. In 2 of these 3 cases there was superficial ulceration of the soft palate, which was slow in healing.

It is commonly believed that paralysis is as likely to follow a mild as a severe attack of diphtheria. In fact, no less an authority than Henoch says :—"Diphtheritic paralysis occurs most commonly after the milder attacks of the disease."[1] So also Dr. Gowers :—"Nor does the severity of the attack of diphtheria determine the occurrence of palsy; it is equally likely to follow attacks that are severe and those that are trifling."[2] With this opinion, however, I do not agree. I will not, of course, deny that paralysis may follow a mild attack, for I have myself seen it do so; but my experience is that the more severe the attack of diphtheria, the greater the likelihood of subsequent paralysis. In diphtheria, severity of attack depends very largely upon extent and persistence of the local exudation—membranous or otherwise. The amount and duration of albumen in the urine give a fair idea of the degree of poisoning by the toxic products of the bacillus diphtheriæ. A reference to the paragraph headed "*Albuminuria*" will show that in, at least, 110 of the 125 cases of paralysis, there was albuminuria, 88·0 per cent.; while in 88 of the cases, 70·4 per cent., the albuminuria lasted for a week or more. Albuminuria was found in about 30 per cent. of the 1,071 cases of diphtheria, amongst which the cases of paralysis under consideration occurred, so that the paralytic cases show a larger proportion having albuminuria. Again, a reference to the table just given will show that in 27, or 21·6 per cent. of the cases of paralysis, there had been little or no local exudation; in 45, or 36·0 per cent., there had been a moderate amount; and in 53, or 42·4 per cent., there had been much; that is, in nearly half the cases of paralysis there was much local exudation, in spite of the fact that it is just the cases with much exudation that are fatal during the attack of diphtheria, at a time too early for paralysis to have supervened.

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[1] "Lectures on Children's Diseases," *Syd. Soc. Trans.*, vol. ii, p. 300.

[2] *Loc. cit.*, p. 904.

Further, I have analysed, in more detail, the severe and the mild cases of paralysis. There were 22 severe cases; 13 of them were, in fact, fatal from the paralysis. In 10 of these 22 cases there was much albumen in the urine; in 10 there was a moderate amount, in 2 there was a trace on one occasion only; and in none was the urine always free from albumen. With the two exceptions mentioned, albumen was present for a period of eleven to one hundred and four days' duration.

With respect to the amount of exudation present during the primary attack of diphtheria in these 22 cases, there was much in 11 cases, a moderate amount in 7, a slight amount in 3, and none in 1. In this last case there was ulceration of the soft palate, which was very slow to heal; and my belief, when I saw the child soon after admission, was that there had been a good deal of membrane before she was sent to the hospital, especially, too, as there was much albumen persisting for at least seventy-seven days. I may also add that there was in 11 out of the 22 cases a discharge from the nose, pointing to the presence of membrane in the nasal passages; and in 5 cases there was croup.

Contrast these figures with the following, which are drawn from an analysis of those cases of paralysis in which only the palate or the ciliary muscles were affected, that is to say, the very mildest cases of paralysis. Excluding 2 fatal cases in which the patients died of the severity of the disease during the primary attack, and in which there was hardly time for the paralysis to have advanced beyond the palate, there were 43 such mild cases. In 7 of these there was much albumen, in 21 there was a trace or a cloud, in 7 there was albumen on one occasion only, and in 8 there was never any albumen. The extremes of times of duration of albuminuria were five and seventy-six days. With respect to the amount of exudation in these 43 cases, the amount was slight in 12, moderate in 18, and much in 13. There was a discharge from the nose in 7 cases, and croup in 3 others.

In a tabular form these figures can be placed as follows:—

Albuminuria.	Severe cases of paralysis.	Mild cases of paralysis.	Faucial exudation.	Severe cases of paralysis.	Mild cases of paralysis.
Much	10	7	Much	11	13
Moderate amount	10	21	Moderate ..	7	18
Present once only	2	7	Slight	3	12
None	0	8	None	1	0
	22	43		22	43

The observations of Cadet de Gassicourt on this subject do not differ very widely from mine. They will be found on page 342 of the third volume of his *Traité Clinique des Maladies de l'Enfance* (deuxième édition), in the admirable chapters which he devotes to diphtheria. De Gassicourt excludes all his cases of diphtheria which were fatal from croup. This leaves him 425 cases, which he divides into three groups: first, the benign cases; second, the severe, toxic and hypertoxic cases; and third, the cases of croup that recovered either with or without operation.

In the first group there were 155 cases with 28 of paralysis, 18 per cent. In the second group there were 135 cases, with 30 of paralysis, 22·2 per cent. In the third group there were 135 cases with 43 of paralysis, 31·8 per cent. The number of fatal paralytic cases in each group was 2, 10 and 8. In a short discussion of these figures de Gassicourt states that he is surprised at the difference between the numbers of cases occurring in the second and third groups; he thinks the explanation to lie in the fact that many of the 135 severe cases of diphtheria died of the malignity of the disease before they had time to become paralytic; but he does not give the actual number who so died.

If de Gassicourt's cases of paralysis are cast into the same form of table as mine it will be seen that out of 101 cases, in 28 the primary attack was benign, in 30 severe, and in 43 there was croup; from the context one gathers that these latter cases were severe or moderately severe cases of faucial, laryngeal and in some instances tracheal diphtheria.

My cases of croup in diphtheria that recovered with or without an operation differ, however, considerably from those enumerated by de Gassicourt. I have had 54 such during the two years 1892 and 1893; 13 of these patients subsequently developed paralysis (24 per cent.), of whom 1 died.

With these facts before us I do not think it can be maintained that paralysis follows a mild attack of diphtheria equally as often as a severe.

The following are brief notes of the 17 fatal cases:—

Case 5.—*Severe diphtheria, faucial and nasal; generalised paralysis; sudden death from cardiac failure.* F., aged 9; sore throat, May 31; admitted on June 5, with the fauces in a very foul condition; there was a sanguineous discharge from the nose, and the child seemed extremely ill. On June 13, the fauces were cleaner; there was still what seemed to be a film of membrane on an ulcerated surface; on the 15th no membrane could be seen, only the ulceration. On June 30, and July 3, membrane was syringed out of the nose. From June 14 to 19th, a well marked papular eruption was to be seen on the skin; on June 22, there was cardiac dilatation; and on July 1, otorrhœa. On this date, too, paralytic symptoms were first observed, the patient being unable to raise her head from the pillow; by July 3, there was marked paresis of the muscles of the trunk and extremities; the voice was nasal; the knee-jerks were absent; the abdominal muscles acted. There was difficulty in swallowing, and paralysis of the ciliary muscles. Pulse 160; respirations 22. The patient remained in the same state till 6.30 p.m., on the same day when she died very suddenly, having been talking and laughing with the nurse almost up to the very moment of death.

Case 6.—*Slight diphtheria, faucial; slight croup, generalised paralysis, including laryngeal and respiratory muscles; death by cardiac failure.* M., aged 5; sore throat, April 19; admitted the next day with a patch of membrane on the left tonsil; by the 27th the membrane had disappeared. There was a cloud of albumen in the urine from April 28 to May 18, and once again on June 2. There was slight croup from April 24 to 27; the patient was troubled with vomiting from April 27 to 30. Soon after he was admitted the cervical glands became enlarged and subsequently they suppurated. The first symptom of paralysis was a nasal voice on May 17. On May 21 there were aphonia, loss of cough (adductor paralysis of larynx), paresis of the arms and legs; no knee-jerks; vomiting; and irregularity of the heart's action. On May 26, he surreptitiously obtained a portion of an orange and nearly choked whilst eating it. On the 30th it was noted that the thorax expanded badly, that the diaphragm was weakened, and that the child could not sit up or even lift his head off the pillow. At 3 a.m., on June 1, there was dyspnœa; the patient became of a bad colour; pulse 120 and regular. At 1 p.m., he was collapsed and there was no dyspnœa; his colour was better. At 11 p.m., he was seized with convulsive movements of the face and arms; the pupils became dilated. These convulsions did not last long. There was some improvement in the patient's condition during the next two days; but he died suddenly from cardiac failure at 5.30 p.m., on June 3.

Case 7.—*Severe attack of diphtheria; paralysis of palate and extremities; death by cardiac failure.* F., aged 4: sore throat on September 8; admitted on September 9, with patches of membrane on the tonsils and fauces; child very ill. There was still membrane on the 11th; the urine contained albumen and continued to do so up till death. The cervical glands were swollen. On October 6, there was a nasal voice. Then the muscles of the extremities became affected. On October 11, there was strabismus; next day there was slight weakness of the diaphragm, and also icterus. The patient died of cardiac failure on October 12.

Case 8.—*Severe diphtheria, faucial and nasal; generalised paralysis; death by cardiac failure.* F., aged 6. Sore throat on July 7; admitted on July 8, with much black tough membrane covering the fauces. By July 13, the fauces were clear. On admission there was rather more than a trace of albumen in the urine; on July 11, the urine was loaded with albumen and it continued to contain much albumen till death. There was also a fœtid discharge from the nose, and cervical lymphadenitis. On July 21, the voice was slightly nasal, the knee-jerks could be obtained but were not well marked, and the pulse 108 and irregular. The patient could read small print. On July 28, the heart was dilated. By August 5, the patient could not stand nor even raise herself in bed. On the 6th, there were no knee-jerks; there was ocular and ciliary palsy. On the 8th, the paralysis was more or less generalised; the patient could not swallow properly. She got worse and died of heart failure on August 11.

Case 9.—*Severe diphtheria, faucial and (?) nasal; generalised paralysis; death by cardiac failure.* M., aged 5. Sore throat on June 4; admitted June 6, with much deposit on the tonsils and palate. The fauces were clear on June 13. There was a discharge from the nose and ears and much swelling of the cervical glands. On June 8, there was rather more than a trace of albumen in the urine, but the quantity increased so that on the 23rd the urine was loaded, and at the same time the amount of urine passed in the twenty-four hours was scanty. This state of affairs continued up till death, except that for a day or two before the end there was a slight improvement in the condition of the urine. Up to June 21, the knee-jerks could occasionally be obtained, but after that date they were absent. Paralysis of the palate was first noticed on June 22; on the 25th there was weakness of the lower extremities. Then gradually paralysis appeared in other parts so that on July 13 there were nasal voice and palsy of the muscles of the upper and lower extremities, the trunk and the neck. On July 14, the patient had to be fed with a tube as he could not swallow. He died the same evening of cardiac failure. As early as June 17, there was evidence of cardiac dilatation; after that date the pulse rate varied from 100 to 124 per minute, and the heart's action was irregular. On the day of death the rate was 172.

Case 10.—*Slight diphtheria, faucial and (?) nasal; paralysis of respiration.* M., aged 3½. Sore throat on February 3; admitted February 7, with membrane on both tonsils. On February 13, the fauces were clear, but on the 14th there was again a small patch on the pharynx. There was albumen in the urine from the day of admission till death. There was also a discharge from the nose, and a cervical abscess which was opened on March 3. Paralysis set in on March 5, with sudden acute abdominal pain; it was observed that the movement of the chest walls was much impaired and the cough was very inefficient, the cause in this case being probably paralysis of the adductors of the larynx. Next day the breathing was almost entirely abdominal; coughing—or rather attempts at coughing—were set up when the child took liquids. In the evening the respirations became of a sighing character and occurred in groups of three with a pause between each group. On March 7, the grasp was feeble; on March 10, the patient could not cough and at times he was of a very bad colour. He died the same day of respiratory paralysis.

Case 11.—*Moderately severe diphtheria, faucial and (?) nasal; paralysis of palate, lower extremities, left external rectus; death from paralysis of respiratory muscles.* M., aged 3. Sore throat January 3; admitted January 6, with tonsils swollen and covered with membrane, which persisted till January 17, and after its disappearance there was ulceration. The urine contained a trace of albumen from January 9 to February 7; and there was a discharge from the nose. On February 8 there was ataxia and some loss of power in the lower extremities; there were no knee-jerks; the palate was slightly affected. On February 9 there was paresis of the left external rectus; February 10, marked nasal voice. February 11, there were attacks of irregular breathing, three respirations taking place in quick succession and then a pause; the pulse was very frequent. On February 13 the character of the breathing had altered; it was quite regular in rhythm, but a long inspiration was followed by a short expiration. In the evening it was noted that there was some paralysis of the muscles of respiration. This breathing continued till death, which took place at 9 a.m. on February 14; the diaphragm became paralysed before death, and there were twitchings of the muscles of the face and arms. Death was due to respiratory paralysis.

Case 12.—*Severe diphtheria, faucial; ocular paralysis; and of upper limbs; death from respiratory palsy.* F., aged 5. Sore throat on July 1; admitted July 5, with much membrane on the fauces, which were clear by July 14. There was rather more than a trace of albumen in the urine; the amount of albumen somewhat increased up to July 14; then it lessened and on August 14 the urine was free from albumen. On July 21 the voice became nasal and there was ciliary paralysis. July 26, patient pale and listless; knee-jerks present. Pulse 88. August 5, squint and diplopia; ciliary paralysis worse. On August 9 the patient could not stand; there was left external strabismus and ptosis on the left side; both the internal recti were weak; the eyeballs did not converge for near vision and there was also loss of the up and down movements. On August 11 the condition was the same, but it was observed in addition that the pupils were moderately dilated; the right reacted feebly to light, but the left did not; neither reacted to accommodation; the upper extremities were partly paralysed. Common sensation was tested and found normal. On August 13 the paresis had increased; the patient could swallow. Pulse 128. August 18, very prostrate. Pulse 80. On August 20 there was frequent cough; and the next day it was noted that there was deficient expansion of the thorax and difficulty in deglutition. The respiratory palsy increased and proved fatal at 1 a.m. on August 22.

Case 13.—*Moderately severe diphtheria, faucial and* (?) *nasal; paralysis of lower extremities, palate and ciliary muscles; death from respiratory paralysis.* F., aged 4. Sore throat May 10; admitted May 11, with deposit on both tonsils; the deposit had increased in amount and become membranous by May 13; it then gradually disappeared and on May 21 the fauces seemed normal. There was much albumen in the urine on admission and up till death. There was also a discharge from the nose. On May 28, it was observed that the knee-jerks were slight and the patient could not stand; pulse frequent; on May 31 the voice was nasal, and next day it was nasal to such a degree that the patient could hardly be understood. On June 3 the ciliary muscles became paralysed; on June 6 there was evidence of slight cardiac dilatation and of weakness of the muscles of the back. On June 15 the heart was still more dilated; there was a systolic-apical murmur and the action was irregular. June 19, heart's action "tumbling" in character; pulse 180. The respirations were very shallow; the patient was unable to cough. In the evening of this day it was noted that there was cyanosis and the patient was "gasping for breath like a newly-caught fish." The diaphragm appeared to be acting, but the intercostal and laryngeal muscles were paralysed; there was still a nasal voice and ciliary paralysis. Pupils dilated. The patient died soon after this note was made.

Case 14.—*Moderately severe attack of diphtheria, faucial and (?) nasal; paralysis of palate and lower extremities; death by cardiac failure.* F., aged 6. Sore throat on May 17; admitted May 19, with a thick covering of membrane on the tonsils and uvula; by the 25th the membrane had disappeared. On admission the urine contained a trace of albumen, which continued up to June 16. The cervical glands were enlarged and there was a discharge from the nose. On June 19 there was considerable difficulty in walking; the voice was slightly nasal; the knee-jerks were not to be obtained, and the heart's action was frequent and irregular. On June 26 the knee-jerks could be obtained. On July 1, in the morning the patient was worse; the heart's action was very frequent and irregular; there was vomiting; no albuminuria. In the afternoon the heart's action was regular, 128; respiration 44; the respirations were laboured, and the colour bad. The child died of cardiac failure at 5 a.m., on July 2.

Case 15.—*Severe diphtheria, faucial and laryngeal; tracheotomy; generalised paralysis; attacks of vomiting and syncope; death by cardiac failure.* M., aged 6. Sore throat on March 19; admitted the same day with much membrane upon the fauces, which increased in amount during the next five days. On March 31, there was much albumen in the urine; the amount then diminished and after April 10 the urine was normal. There was croup which necessitated tracheotomy on March 24. From this the patient completely recovered, and was allowed to get up on April 10. On April 29, there was paralysis of the palate and weakness of the right arm; the knee-jerks could not be obtained. Afterwards there was more or less generalised paresis; on May 3, attacks of vomiting and syncope commenced which recurred occasionally during the next few days and in one of which the patient expired early in the morning of May 8.

Case 16.—*Moderately severe diphtheria, faucial; nephritis; paralysis of palate, extremities and larynx; constant vomiting; death by cardiac failure.* F., aged 42. Sore throat June 28; admitted July 3; with much membrane on the fauces. The membrane had gone by July 8, but there was ulceration of the soft palate and uvula which did not heal up till July 23. It was not possible to examine the urine till July 6, and it was then found to be loaded with albumen and to be smoky, which it continued to be up to July 11. There were also casts. The condition of the urine pointed to an acute nephritis. After July 20, the amount of albumen lessened, and on July 27 there was rather more than a trace, which continued; on August 29 there was a faint trace. From July 13 to 18th the patient was troubled with pain in the chest, to account for which no physical signs were detected; there was also occasional vomiting; pulse 60 to 64; heart intermittent. On July 31 there was a return of fluids through the nose; on August 6 the voice became nasal; on the 19th there was paresis of the upper and lower limbs and ataxia; no jerks could be obtained either in the upper or lower extremities. August 25, vomiting; cough after taking food; pulse 96. During the following night there was vomiting and abdominal pain with collapse, all coming on rather suddenly. Pulse 90; the action and sounds of the heart were normal the next morning. The patient was fed by nutrient enemata on account of the constant vomiting. On August 27 she was almost voiceless; there was frequent vomiting without any feeling of nausea; pulse 96; regular. On August 28 the patient was quite helpless; there was almost complete loss of power in the lower, and marked paresis of the upper extremities; vomiting continued. Pulse 96; heart normal and regular. The patient got weaker and died quietly shortly before 6 a.m., on August 29. Only a partial *post-mortem* examination was permitted. The abdominal organs were found to be quite normal to the naked eye.

Case 17.—*Severe diphtheria, faucial and nasal; paralysis of palate, intercostals and laryngeal muscles; death in convulsions.* M., aged 4. Sore throat February 15; admitted February 17 with much membrane spread over the fauces. Membrane was still present on February 24; on February 28 there was a trace of albumen in the urine; there was a discharge from the nose; later also diarrhœa and a cervical abscess. On March 13 there was paralysis of the palate; on the 15th there was weakness of the intercostals and adductors of the larynx; the patient was seized with convulsions during the day and speedily died.

Case 18.—*Severe diphtheria, faucial and nasal; paralysis of palate; death from severity of attack.* F., aged 10. Sore throat November 13; admitted November 15, with very much membrane on the fauces, which had much decreased but not quite disappeared at the time of death. There was albuminuria, and a discharge from the nose. The patient was very ill. On November 24 there was a decidedly nasal voice; heart normal. The patient got weaker, and died from the effects of the poison of diphtheria on November 28.

Case 19.—*Severe diphtheria, faucial and nasal; paralysis of palate; death from severity of disease.* M., aged 9. Sore throat on May 10; admitted May 13 with much swelling of the fauces and much membrane. The membrane gradually disappeared by May 24. There was a good deal of albumen in the urine from the day of admission to the day of death. There was epistaxis on the 16th and 17th. On May 23 the patient had a nasal voice. No further symptom of paralysis occurred till the morning of May 29, when there were symptoms of cardiac failure; no vomiting. The patient died of cardiac failure at 7.30 p.m. the same evening.

Case 20.—*Moderately severe diphtheria, faucial with slight croup; generalised paralysis; relapse of diphtheria, faucial and laryngeal; tracheotomy, death.* F., aged 2½. Sore throat on September 25; admitted on September 28 with membrane on the tonsils and uvula; there was still a little membrane on the uvula on September 30; there was a cloud of albumen in the urine on September 30; albuminuria existed till November 20. When the patient was admitted there was slight croup. Paralysis of the palate was observed on October 6; by November 11 there was more or less generalised paresis with strabismus. The child however had much improved when on December 28 a relapse of diphtheria occurred; the membranous formation spread to the larynx and trachea; the child was tracheotomised with only temporary relief, and death took place on January 2.

Case 21.—*Prolonged diphtheria; faucial, laryngeal and nasal; paralysis of palate, ciliary muscle and one external rectus; death from direct effects of diphtheritic poison.* M., aged 6. Admitted July 30; there was swelling of the tonsils, with membrane on the left tonsil and on the uvula. On August 1 there was no membrane but slight ulceration in its place. On August 9 there were again some specks of deposit on the fauces; these cleared off, but again on the 20th the fauces became swollen, and there was membrane on the right tonsil; the next day there was some on the left as well. On August 25 there was a discharge from the nose and enlargement of the glands in the neck; on the 26th slight croup appeared, and next day the child coughed up a small piece of membrane There was still membrane on both tonsils on August 31. There was a cloud of albumen in the urine when the patient was admitted; the amount quickly increased, and for some days before death the urine was loaded. Symptoms of paralysis set in on August 3 in the palate, the ciliary muscles being also slightly affected. On August 6, 15 and 19, it was noticed that the knee-jerks were exaggerated; by the last date the palsy of the palate and ciliary muscle had become more pronounced. On August 23 the knee-jerks had almost disappeared. On September 5 the left external rectus was paralysed. The patient died on September 6, most probably from the direct effects of the diphtheritic poison.

An analysis of the 13 cases which were fatal from paralysis shows that in them the local exudation persisted for periods varying from six to thirty-three days. The onset of the paralytic symptoms was observed on from the fourteenth day at the earliest to the forty-first at the latest, counting from the commencement of the attack of diphtheria; while the fatal event occurred at from the third to the thirty-fourth day from the commencement of the palsy.

Onset of Paralysis.		Date of death, from onset of paralysis.	
First week ..	.. 0	Within one week ..	.. 4
Second week..	.. 1 (14th day)	„ two weeks ..	.. 3
Third week ..	.. 3	„ three weeks ..	.. 2
Fourth week..	.. 3	„ four weeks ..	.. 2
Fifth week ..	.. 4	„ five weeks ..	.. 2
Sixth week ..	.. 2		13
	13		

Cadet de Gassicourt in the treatise I have already cited divides cases of diphtheritic paralysis into three classes. According to this arrangement the paralysis is (1) *limited;* (2) *generalised,* or (3) of the *cardio-pulmonary* form. Cases of the first class are those in which but one or two muscles or groups of muscles are affected. De Gassicourt states that it is rare for the paralysis to be limited to any part other than the palate and pharynx, and that this particular form of limited paralysis forms a very large proportion of all cases of paralysis; in fact he states that out of 128 cases of diphtheritic paralysis observed by himself in 103 the paralysis was so limited. My experience has not been the same as his in this respect, as will be seen on reference to the paragraph of this paper headed "extent of paralysis." Nor do I find that my cases fall readily into these three divisions. There are many cases which are intermediate as regards extent of paralysis between the limited and the generalised forms.

The large majority of cases of limited paralysis terminate in recovery. But, as in Case 10, the paralysis may fall upon an important group of muscles, those of respiration for instance, and so prove fatal.

Of the generalised form of paralysis in varying degrees, Cases 5, 6, 8, 9, 13, 15, 16 and 20 are examples. Of severe cases of recovery from generalised paralysis the following are good instances.

Case 22.—M., aged 3. Sore throat on June 7; admitted the same day with membrane on both tonsils, which had cleared off by June 18; there was a cloud of albumen in the urine; the amount of albumen increased so that on July 1 there was much; then it diminished, and after August 1 the urine was normal. On June 21 it was noted that the pulse was 108, and the heart's action at times very irregular. Next day there was a nasal voice and return of fluids through the nose. The condition remained much the same for some days, but during the first week in July the legs became affected and by the 8th the child was unable to stand, though he could move his legs in bed; the knee-jerks were lost. On July 17 paraplegia was complete; on the 18th there was inability to swallow and the heart was irregular; on the 21st the patient could not lift his head from the pillow, and on the 25th he was unable to turn himself in bed and, in fact, to help himself in any way, the muscles of the upper and lower extremities, trunk and neck, being paralysed. On July 26 the diaphragm became affected; the intercostals, however, seemed to be acting well; there was also inability to cough; on the 28th both external recti were palsied; on the 31st the pulse was 130 and regular, respiration 30; the face was continually bathed in perspiration; the diaphragm acted but slightly; the patient could only make attempts to talk; it was with difficulty that he made even a sound; one could see him make an inspiratory effort and then try to utter a syllable, each syllable requiring a fresh effort.

This state of things remained till August 5; it was then noted in addition that the saliva dribbled from his mouth. After that date, however, he began rapidly to improve; on August 10 he could swallow; by the end of the month he could sit up and during the first week in September was able to walk. He was discharged well on September 20. This is the most severe case of generalised paralysis that I have seen recover.

Case 23.—F., aged 5. Said to have first had a sore throat on April 10, but was not admitted till the 21st; there was membrane on each tonsil and a cloud of albumen in the urine, which continued up to May 29, after which date the urine was normal. There was also croup which necessitated tracheotomy on April 23; a good deal of membrane was coughed up through the tube up to the night of April 26; on April 29 the tube was finally removed; by May 29 the wound was quite healed; on this date the voice was noticed to be nasal; in the afternoon the child became collapsed and was unable to cough properly, probably because the vocal cords could not be approximated; there was pallor; pulse 102. On June 2 the pulse was 120; it had previously been 160; the child could not sit up or lift her head off the pillow. There was paresis of the left external rectus. On June 6 there were attacks of collapse and frequent coughing; the wound had broken down; (it did not heal up again thoroughly till July 17). On June 9 there was decided paralysis of the upper and lower extremities; the abdomen did not move at all in respiration, but the abdominal muscles could be felt to act when the patient tried to cough. Breathing was chiefly performed by intercostals. June 21, cough still aphonic. July 3, spoke to-day for the first time since the beginning of June in a fairly loud voice, which is slightly nasal; no squint; can swallow; paralysis of limbs better. On July 9 in the morning there was an attack of vomiting, with prostration and coldness of the extremities; at noon the pulse was 146; at 11 p.m., 90 and occasionally intermittent. July 10, pulse 100, regular. July 17, pulse 96, regular; voice still slightly nasal; otherwise the patient's condition has much improved; she can sit up but not stand. July 27, can walk by herself but is unsteady. Went home quite well on August 12.

But by far the most interesting group is the third, the cases of cardio-pulmonary paralysis (de Gassicourt), the cardiac and vagus paralysis of other writers. From what I understand, authors describe more than one class of case under the head of cardiac paralysis. Henoch, for instance, says that "risk of sudden death from failure of the heart's action hangs over the convalescent like the sword of Damocles;"[1] and he instances cases in which somewhat sudden death took place from cardiac failure, three or four weeks after the commencement of the attack of diphtheria; there having been no other paralysis. Again Dr. Goodhart writes, "but in calling diphtheritic paralysis an affection of the convalescent, we must not forget that in the active stages of the disease we have also a paralysis which constitutes one of the gravest dangers of diphtheria—viz., paralysis of the heart; one can but suppose, indeed, that this is a part of the same tendency to the implication of the nervous centres as is seen in the stage of convalescence, and that in those terrible cases of sudden death, which are by no means uncommon both during the disease and convalescence, we have some sudden disturbance of the vagus, which thus manifests itself by means of its cardiac branches."[2] Further on in his book Henoch says: "the important connection between the nervous system and the production of this cardiac paralysis, seems to me to be indicated by the fact that these cardiac symptoms not uncommonly set in in the course of diphtheritic paralysis, even in ordinary cases of paralysis of the palate only, and cause death from syncope almost at once."

Now it seems to me that the pathology of cardiac paralysis may be different in different cases of diphtheria. First of all we have those cases in which syncope, which may prove fatal, occurs during the acute stage of the attack of diphtheria—perhaps it would be better to say during the time when exudation is still present upon the fauces or on one of the mucous membranes,—and without there being any symptom of any other paralysis (palate, ciliary, &c.). Secondly, there are the cases in which the patient having already become more or less paralysed is seized with symptoms of cardiac failure, which may and often do rapidly end fatally. Thirdly, there is the group in which the patient, being convalescent and having been free from membrane for some days or even three or four weeks, is seized with symptoms of heart-failure which also may terminate in speedy death.

Cases of the first description are so common that it is unnecessary for me to say anything further about them from the clinical point of view. Some cases of diphtheria frequently die of cardiac failure, or "cardiac paralysis," at times suddenly; at other times slowly. But the cause of this heart-failure is rather to be sought in the immediate action of toxic products of the bacillus diphtheriæ on some part of the nervous mechanism, than in actual changes of a degenerative nature in the nerves or muscles. Death in this manner is also seen in scarlatina maligna, and in that disease we do not hear of "paralysis." Cases 18 and 19 are examples in my opinion of this mode of death, even though in both of them paralysis of the palate had supervened; both patients were very ill before this event and were in danger of death from heart-failure quite apart from the occurrence of any other paralysis. In Case 21 also, the patient was profoundly poisoned before the onset of the paralytic symptoms. If these cases of death from cardiac failure during the presence or very soon after the disappearance of membrane are to be classed amongst cases of diphtheritic paralysis, then the numbers of paralytic cases would be much increased and the death-rate (from paralysis) be much higher. This group of cases should therefore be separated from the other two.

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[1] *Loc. supra cit*, vol. ii., p. 296 [2] "Diseases of Children, 4th ed., p. 204"

Of cases in which cardiac failure supervenes upon a more or less extensive paralysis, Cases 5 to 9 are examples. In four of these cases evidence of cardiac disturbance was observed during the course of the paralysis, and in three (Cases 5, 8 and 9) there was cardiac dilatation. In these cases heart-failure may have been due to disordered innervation or simply to fatty changes such as we know take place in the cardiac muscle. In those of the cases where there was dilatation these changes may well have been the cause both of dilatation and failure of the heart.

There remains to be considered the third group of cases, namely those in which death more or less sudden from cardiac failure, occurs during the stage of convalescence. I have no doubt myself that some of these cases are due to a degeneration of the cardiac muscle, and that others are due to a degeneration of the vagus or its branches. The latter cases are much less common than the former, and it is of these that I wish especially to speak.

The symptoms usually associated with an affection of the vagus are (1) alteration in the action of the heart; (2) paresis or paralysis of the intrinsic muscles of the larynx, and alteration in the rhythm of respiration; and (3) vomiting. Examples of paralysis in which these symptoms were present in varying degree will be found among the cases previously detailed, especially Cases 6, 14, 15, 16 and 23; in the last case there was recovery. In Case 16, symptoms of cardiac failure with vomiting and aphonia supervened some days after the onset of a generalised palsy, and rapidly proved fatal. But, as I have said, there was evidence in these cases of nerve degeneration, shown by the paralysis of the limbs, &c. In the following case these symptoms (cardiac, respiratory and gastric) were present and proved fatal, quite apart from any other paralysis. The case occurred during the summer of 1892, but in consequence of the uncertainty that exists as to whether these symptoms are really due to vagus-degeneration or not, it has not been included in the statistics of paralysis given in the early portion of this paper.

Case 24.—*Severe diphtheria, faucial and nasal; cervical abscesses; (?) palsy of vagus.* M., aged 4½ years; admitted on May 10, with a severe attack of diphtheria which had commenced on May 8; there was membrane on both tonsils, a muco-purulent discharge from the nose and much cervical glandular enlargement. The patient was restless but took nourishment well; pulse 142; temperature 102°·8 F. The urine contained albumen. By May 13 the deposit (membrane) had cleared off. The boy died on the evening of May 27, and the symptoms may be stated briefly as follows:—He gradually became weaker but never paralysed anywhere; there was albumen in the urine in variable amount; on May 25 the urine became rather scanty; the pulse remained persistently frequent, 130 to 140; there was much pallor all along, for the last three or four days extreme pallor; on May 23 the heart was slightly irregular and so also was the respiration; the patient took a dozen or so deep breaths and then there was a period of apnœa; there was also occasional vomiting during this day; slight stridor on inspiration with some recession of the six lower spaces and epigastrium; this continued more or less up to the end. On the 24th the reporter notes "the respiration is very irregular; the child appears to forget to breathe and afterwards to relieve himself by deep-drawn, laboured inspirations which are not followed by the proper amount of chest movement." Pulse 132 and very feeble. On the 24th for the first time there was wandering in mind, though the patient was quite sensible when spoken to. Temperature 96·8° F. On the 25th the apex of the heart could be seen beating violently one inch external to the nipple line. There was vomiting during the night. The child died quietly, the symptoms remaining much the same up to the end. The cervical glands had got a good deal larger and were found *post-mortem* to contain pus tensely encysted by firm tough capsules; otherwise there was nothing amiss, to the naked eye at any rate.

Here we have a patient, who, on about the sixteenth day from the onset of a severe attack of diphtheria, and about ten days after the local exudation has disappeared, is attacked with (1) cardiac disturbance; (2) respiratory disturbance; and (3) vomiting; with which symptoms he dies in five days, there being no other definite paralysis.

There is no *a priori* reason why one or both vagi should not be the first nerves to undergo degenerative changes in any particular instance; and it is easy to understand that if such important nerves are early affected, death may ensue before sufficient time has elapsed to afford clinical evidence of degeneration of other nerves. Such cases as the one just related are, however, very uncommon.

CASE OF CEREBRAL TUMOUR IN WHICH THE INITIAL SYMPTOMS WERE CHIEFLY SENSORY IN THE ARM AND FACE. SUDDEN AGGRAVATION WITH LOSS OF HEARING IN CORRESPONDING EAR.

Post-Mortem.—Tumour found in White Substance chiefly involving Posterior Part of Internal Capsule.

BY SAMUEL WEST, M.D., F.R.C.P.

Assistant Physician, &c., to St. Bartholomew's Hospital; Senior Physician to Royal Fever Hospital, &c.

Percy B., aged 16, was admitted into John ward on January 3, 1895. He had been in good health until six months ago when he had a blow on the side of the head with a pillow. He immediately felt dizzy, could not speak plainly, and had a feeling of numbness in the fingers of the left hand, a peculiar feeling in his face, and he said he could not laugh properly. He had no pain, did not vomit, and slept well afterwards. Ever since then he has had pain in the head, has felt weak in the left hand, has been liable to drop things, and has had numbness in the finger and palm. Occasionally a sudden pain would shoot down the arm, causing it to jerk irregularly; more rarely down the left leg, causing it to give way. He could walk and run well, could stand with his eyes shut, was not giddy, and was quite able to do his work until admission.

About three months ago his sight began to get a little weak, but it was not till a month ago that it became misty. Headache he had all his life long, but it had been worse since the blow, and has been felt recently in the frontal region, especially on the right side, and has been aggravated by cough or exertion. Five months ago he began to be troubled with vomiting, which has recurred on and off ever since.

On examination the lad looked well, and there was really nothing to note, except that there was slight weakness in the left hand, but very little in the grasp, and that the sensation over the left arm in the hand and palm was distinctly impaired, but only very little. On examining the eyes there was found to be well marked double optic neuritis. Both discs were enormously swollen, but the swelling was in excess of effusion, for the vessels were tolerably distinct all over, and there were no patches on the retina. It was the condition which is usually described as "swollen discs." It was thought that the sensation in the left leg, and also in the left side of the face, was slightly impaired too, but it was very indefinite. The epigastric, abdominal and cremasteric reflexes were all weak on the left side but active on the right. The knee-jerk was absent on both sides.

What brought him into the hospital was his loss of sight and the peculiar sensations in his arm. He did not seem seriously ill on admission.

The diagnosis was made of cerebral tumour, possibly, it was thought, of a tubercular nature. A few days later he became very much worse, had more headache, vomited several times, and was more drowsy and apathetic. Some weakness was observed in the leg, and the sensation was distinctly impaired there, while the weakness of the left hand and the loss of sensation were more marked. At the same time the movements of the left side of the face were distinctly impaired, and the sensation also was obviously defective. The pupils, which had been equal throughout, reacted now sluggishly to light as well as to accommodation, but remained equal. On January 17 the patient was very much more drowsy and had passed water under him. The hearing was noted now to be defective. He could only hear a watch one inch away with the left ear, while the hearing on the right side was fairly acute. The other symptoms were much the same though slightly aggravated.

The diagnosis having been made of cerebral tumour, and the father, when he was informed that no surgical operation could be performed, nor any special treatment recommended, decided to take the patient home, where two days later he died.

A *post-mortem* examination was made.

The interest of the case lay in the diagnosis of the seat of the new growth. From the predominance of sensory symptoms from the first, which were more or less *hemiplegic in type*, it seemed that the tumour must exist in the white substance of the brain, and probably in such a position as to interfere with the posterior portion of the internal capsule. In this connection it was very interesting to note the loss of hearing which developed towards the end of the case at the same time as the *hemiplegic* symptoms became more marked. On opening the skull the right side of the brain was noticed to be more prominent than the left, but no tumour was visible on the surface. On section, a tumour was found as large as a walnut, lying in the white matter to the outer side of the lenticular nucleus.

The bulk of the tumour was above the level of the *basal ganglia.* The lower and posterior part was surrounded by an area of softening which involved the whole of the external, and the hind extremity of the internal capsule, and extended backwards into the occipital lobe and downwards somewhat into the temporo-sphenoidal lobe. There was some small hæmorrhage in and around the tumour, the whole brain on that side being softer and more watery than normal. The tumour proved to be a *glio-sarcoma.*

Its seat fairly well explained the symptoms observed during life. The vision we might have expected to be different on the two sides, but the fields of vision were carefully examined during life and were only found to be symmetrically reduced. I suppose that the tumour must have existed for some time, and that the recent symptoms must have been due to the development of the softening, which proved to be so extensive on *post-mortem* examination.

A CASE OF UNILATERAL COMPLETE OPHTHALMOPLEGIA.

BY FRED. TRESILIAN, M.D., M.S.

Thomas H., aged 43. When I first saw him (April 3) he complained of general malaise, and said he felt as if he had a feverish cold. During its progress, however, he developed a convergent squint in the right eye, and examination showed complete paralysis of the external rectus, the eye making no movement whatever outwards. He then had obstinate pain in the right side of the head, especially over the temple, and around the eye, which was especially severe at night, and prevented him from getting any sleep, except an occasional nap during the day. He also had frequently sudden and unexpected vomiting not occurring after meals, but when it did, any food present in the stomach was rejected. After a few days ptosis appeared, which soon became complete, and very rapidly all the ocular muscles were paralysed. The eye was now perfectly immovable, and directed straight forwards, and he could not raise the lid. There was slight proptosis, and the pupil was more contracted than the other, and acted very feebly to light. Vision, however, was very fair, but much worse than in the other eye. An ophthalmoscopic examination, which had to be done with a candle, showed only indistinctness of the margins of the disc. He had a chancre followed by a sore throat, and a skin rash, eight years previously.

There was a large sore on the scalp, over the posterior superior angle of the parietal bone; this discharged pus, and was connected by an opening with the cranial bone. There were also two small discharging sores on the sole of the left foot. These three must have been gummata. He complained of a feeling of deadness in the skin over the eyebrow, and I found there was anæsthesia, though not on the nose, nor below the orbit. Knee-jerks normal.

Two days afterwards. Pupil did not act to either light or accommodation, and its size was 3½ m. About two months later there was again an internal squint in the affected eye, and there was some power of movement upwards and downwards. Some condition of episcleritis was present. I found the optic disc normal. Pupil reflex still lost, accommodation also. V. O. S. normal, O.D. = counting fingers. The sores on the head and foot were quite healed. The ptosis was not quite so much. In September there was still some internal squint, the ptosis had disappeared, the pupil acted to both light and accommodation. The vision was much better. After this I lost sight of him. My diagnosis was a gumma at the back of the orbit, about the region of the sphenoidal fissure, and implicating the various ocular nerves as they entered the orbit. The treatment was pot. iod. gr. 20, t.d.s. The gummata were dressed with iodoform ointment.

ALCOHOLIC PERIPHERAL NEURITIS IN ADVANCED AGE.

BY ARTHUR MAUDE.

Mr. C., a robust country gentleman of sporting outdoor habits had taken stimulants to excess for certainly twenty years. There was good evidence that some eighteen years ago no unusual daily allowance was half a gallon of beer, a bottle of sherry and perhaps eight or ten liqueur glasses of neat whiskey. In spite of ample means his favourite drink was beer, and he would in his later years sit drinking beer all day, beginning at breakfast. During the summer of 1894, when over 75 years old, he would often consume two quarts of beer, a bottle of sherry and half a bottle of whiskey in one day. This large amount of alcohol never seemed to disturb his digestion or very greatly affect his appetite; and many years ago, his relatives assert, he would sit down to dinner apparently quite drunk, eat a good meal and get up "quite sober." He never had any serious illness, except broken bones from riding accidents. He "carried his liquor" in such a remarkable way that in 1892 he fell downstairs one night going to bed, but contrived to undress himself and get to bed without discovering (as his valet did next morning) that he had broken his humerus. Excellent union resulted with no impairment of the shoulder joint (under the writer's care).

For several years he had presented increasing hebetude of mind, but not to an undue degree for his age, while his memory remained excellent when roused. Even after such a severe accident he showed no signs of delirium tremens. During the summer of 1894 his general muscular power became greatly diminished, so that he could only just stand and walk with assistance. In October, 1894, he began to complain of darting pain of a severe character over the distribution of the left posterior tibial nerve. About the middle of November the hands and feet began to swell rather suddenly, while a few weeks before there were no signs of œdema.

December 7.—The hands are much swollen, the backs being quite tense, and shiny, the skin exceedingly thin and glossy; there is no swelling of the fingers and he can remove his rings easily. The skin of both hands is much discoloured and livid, with small, defined ecchymoses dotted over them.

The insteps of both feet present the same condition. There is œdema of calves and thighs, while the muscles are much shrunken. The face is puffy and bloated, but there is no œdema of the eyelids.

There has been some tendency to spontaneous ecchymoses for several years. He can just stand with assistance and shuffle a few steps with feet wide apart. There is, however, no definite extensor paresis. Tendon reflexes (patellar) quite absent on both sides (tested by Buzzard's method). Pupils contracted to pin points, no reaction to light or accommodation.

Urine 1,020, highly acid, dark from bile pigments, no albumen, no glycosuria.

Heart—feeble in action, regular, no sign of dilatation. Arteries soft for his age. Great dyspnœa on the least exertion.

No gastric or intestinal disturbance; liver not apparently enlarged.

No hyperæsthesiæ; no pain except in the left leg and thighs. No deafness.

Can move his fingers naturally and oppose his thumbs and little fingers properly.

December 10.—Both radial pulses were of fair volume, though feeble in action.

December 11.—Both pulses were found almost impalpable, though the carotid pulse was good, and the heart sounds were not more feeble.

December 12.—The fingers of the right hand, especially the two middle ones were quite blue and livid, but not swollen. The œdema elsewhere is distinctly less, probably owing to diuretics and digitalis with strychnine that he has been taking.

December 13.—Died quite suddenly after a few moments' dyspnœa.

No *post-mortem* allowed.

Remarks.—The peculiar nature of the œdema and the symptoms of local asphyxia leave no room for doubt that the condition was one of peripheral neuritis. If so the case is worthy of record from the extreme age of the patient, as it is very rarely that a man with such habits survives long enough to manifest such symptoms at 75. The continued habit of beer drinking bears out Dr. Ross's observation on the point ("Peripheral Neuritis," p. 150). The intensity of the vasomotor symptoms as compared with the motor disturbance is striking. Contracted fixed pupils have been noted by Fischer (Ross, "Peripheral Neuritis," p. 155).

The trophic phenomena were well marked, viz., the thin, smooth, glossy skin, and dry, brittle nails, but they were probably exaggerated by old age. About a year before death there had been an irruption of "papular eczema" limited to the extensor surfaces of both arms and the nape of the neck and interscapular regions (Ross, p. 158).

The purpuric spots are also a condition found in old age; it is noticeable that they occurred more frequently and were of larger area in the right hand, possibly because the nerve trunks of this arm had been damaged by the fracture two years before.

The absence of all distinct mental change is remarkable, but it must be remembered that this gentleman had always been in easy circumstances and for years had led the life of an absolute recluse, scarcely seeing anyone except his own servants, and for several years had never left his house and gardens.

ON IMPERATIVE IDEAS.[1]

Being a Discussion on Dr. Hack Tuke's Paper (BRAIN, 1894).

1.—DR. HUGHLINGS-JACKSON.

ALTHOUGH I had not the pleasure of hearing Dr. Hack Tuke's very important paper I have read it and beg to offer some comments upon it. After its perusal I find that I have several times seen cases in which there were what Dr. Tuke calls "imperative ideas." Unfortunately I paid but little attention to these cases in a scientific regard and certainly did not view them in the wide aspect Dr. Tuke so ably presents them. From his researches on this curious and important matter I feel sure neurologists will profit greatly. One thing which impresses me strongly is that Dr. Tuke deals with the cases of morbid ideation, he mentions as they are departures from normal mental states or at least as they are exaggerations and persistencies of mental states which at their origin it would be pedantic to call abnormal.

In the Leeds address (*British Medical Journal*, vol. i., 1889) to which Dr. Tuke refers I have suggested that certain very absurd and persisting delusions are owing to fixation of grotesque fancies of dreams in cases where a morbid change in the brain happens suddenly, or when one increases suddenly, during sleep. I will not apply this hypothesis without qualification to such cases as Dr. Tuke relates. It may, however, be that certain obtrusive thoughts which otherwise might be transitory become fixed, become "imperative ideas," consequent on some very slight morbid change in the brain occurring during sleep. I suppose indeed that Dr. Tuke means that a normal mental state, or at least one which is very common in persons taken to be quite healthy, becomes more or less fixed when general health is lowered or when some quasi-trifling morbid change occurs in the brain itself. Dr. Tuke writes under the head of "Dread of Dirt": "Should the general mental power be lowered, or a hereditary predisposition exist, this tendency to attend to the state of the skin becomes a passion, and results in the needless and wearisome washings of the body or the dress. Indeed it too often happens that the patient no longer perceives the folly of these ablutions, but labours under an actual delusion." We certainly have to account for the existence of these quasi-parasitical states in cases where the general mental power is but little lessened. The following remarks Dr. Tuke makes are to this point and illustrate what I said on his regarding "imperative ideas" as departures from normal or nearly normal mental states: (I italicise some words) "What strikes one forcibly in regard to most, if not all, imperative ideas is that between them and ordinary ideas *the difference is one of degree*, and that it is a most difficult thing to determine when the boundary line has been passed. This on the psychological side. And on the physical side, what we want above all things to know is what cerebral change, degeneration, or dissolution has occurred by which *the man who to-day is conscious of and smiles at an imperative idea, gravely believes it to-morrow and labours under a systematised delusion.*" Further on, Dr. Tuke writes: "The predominating idea has, in fact, become part of his [the patient's] own being." It is then a quasi-parasitical mental state of the patient, as indeed Dr. Tuke's words "predominating idea" mean.

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[1] Since these articles were received by the Editor, we have had to deplore the untimely death of Dr. Hack Tuke, who was intending to sum up the discussion in replying to some of the criticisms.

In the Leeds address already referred to I have spoken of "fixation of states" in suddenly occurring lesions in cases which are not insanities; aphasia for one example.

I think Dr. Tuke has done good service in drawing attention not only to the long past researches of Laycock, but to important principles more recently stated by Anstie and Thompson Dickson and by Monro. Monro, so far as I know, first enunciated the doctrine that in cases of insanity the abnormal mental condition is double, that in it are two opposite elements, one negative and one positive. [Of course there is the obvious exception of complete dementia (deepest dissolution) where the mental condition is entirely negative.] For my part I consider that illusions, delusions and all other positive mental symptoms in insanities signify activities of healthy nervous arrangements of the highest cerebral centres (so-called "organ of mind"). What we call an insane man's illusions are his perceptions, what we call his delusions are his beliefs, and more generally his positive mental symptoms sample the mentation remaining possible to him, a mentation occurring during activities of what is left intact of his highest centres, of what disease has spared. The physical condition for these positive mental symptoms is not caused, using the word cause in its strict scientific sense, by disease, not caused, that is, by a *pathological process*. Disease is, I submit, answerable only for the co-existing negative mental element of insanity. Here let me remark that, to take one kind of mental symptom, an illusion, a positive mental state implies a co-existing negative mental state; if a man sees a black cat where there is only a black felt hat, not only *is there for him* a black cat, but *this is not for him* a felt hat. Similarly, *mutatis mutandis*, for other positive mental symptoms sampling the positive element of a patient's insanity. As to the physical. Disease of the highest range of the highest centres producing loss of its function or destroying it, answers to the negative mental element in a case of insanity; whilst the activities of the lower, intact, ranges answer to the positive mental element. But sometimes the positive symptoms are super-positive as in post-epileptic unconsciousness (negative element) with mania (positive element). In these cases the lower ranges are in over-activity, and yet I think that these ranges are healthy, that they are untouched by the pathological process which has produced loss of function of, or has destroyed, the highest range. Their over-activity is an hyper-*physiological* state and is analogous to the heart's beating more frequently after section of the vagi. Here comes the application of Anstie's principle. The loss of function or destruction of the highest range of the highest centres is *not only a loss* (answering to the negative mental element of an insanity), but is also a taking off of control from the lower ranges; the "taking off" of the higher is a "letting go" of the lower. The disease may be said to be "constructively accountable" for the over-activity of the lower ranges, for, had not disease destroyed the highest range, that highest range would have continued to control the lower ranges.

I have suggested that the *rate* with which control is removed is an important factor in insanities—that the lower ranges are, at least temporarily, in greater activity the more rapidly the highest range is rendered functionless or destroyed. When disease of the highest range progresses very slowly there may be no obtrusive positive mental symptoms (control slowly removed); when it is very rapid the patient's mentation (the mentation remaining possible to him) is at a great rate (control rapidly removed).[1]

With regard to what Dr. Tuke says in his first paragraph I submit that the scientific study of insanities may be best begun in general hospitals. For in them we encounter cases in which there are very slight departures from the patient's normal mental state; the slighter departures are most easily analysed. I have tried to illustrate this ("Neurological Fragments," *British Medical Journal*, March 5, 1892). We see in these hospitals, however, all degrees of departure from normal states from slightest temporary confusion of thought to deep coma. We should study cases of departure from normal mental state regardless whether or not they are cases of insanity ordinarily so-called, delirium in acute non-cerebral diseases, pneumonia for one example. We should, I think, find in the degrees of insanities so occurring some index of the general condition of the patients and thus some guide to treatment of their cases. I think we should learn something of great value from the analytical investigation of the abnormal mental condition in certain cases of poisoning by cannabis indica and belladonna. Dr. H. C. Wood in his work "On Therapeutics" records his experiences after taking a large dose of cannabis americana. A careful study of the abnormal mental condition this distinguished physician records will, I submit, in a scientific regard, be more profitable than that of most cases of insanity ordinarily so-called. Among other things there was "the sense of prolongation of time," a subject of great psychological interest; this "prolongation of time" occurs in dreams and in some cases of ordinary delirium.

In some cases of intracranial tumour there is a general lowering of mind or synonymously of consciousness towards and sometimes close upon complete dementia. I fear, however, we do not give enough attention to mental failure in these cases when it is slight. The degree of mental degradation will, I think, be helpful in gauging the patient's condition, especially with regard to the degree of raised intracranial pressure from the tumour. Here is a most important practical matter and one of pressing interest now that Victor Horsley has, so to speak, put the question of raised intracranial pressure on a new and definite basis with regard to several important symptoms of brain disease. I dare say it might be said of such cases where there was only much hebetude or great obtuseness that there were no mental symptoms. This would be rather careless since hebetude and obtuseness are in this application words synonymous with defect of consciousness (negative mental state). In these cases the negative mental condition is most or solely marked; positive mental *symptoms* are rare, and I suppose this happens because intracranial pressure is slowly raised, the brain slowly squeezed; hence the functionable remainder of the brain will be in but little or in no over-activity; control being slowly removed.

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[1] I do not go into detail here as I have recently (June, 1894) published a paper in the *Medical Press and Circular* on "The Factors of Insanities"

2.—Dr. G. H. Savage.

After hearing the full and very suggestive paper read by Dr. Hack Tuke I feel that what I have to say must be rather in the way of complement to what has been so well said, while I also give the more special experience which I have had during the past twenty years. First of all, the term "imperative" ideas in one respect is misleading, for though these ideas are at times compelling they are not always the source or spring of action; they command in a way, but to command rather infers that they expect some result from the command; whereas there are certain patients who have had dominant ideas for years who have never felt that these had any relation to their actions or their conduct. Some imperative ideas are suggestive and others compelling.

As to their causation in my experience the most common predisposing cause has been hereditary predisposition to some kind, some neurosis or a tendency to nervous degeneration present in the patient's parents or other marked evidence of nervous instability in brothers, sisters or cousins.

As to exciting causes, or if I may use the term, the determinant of the form of imperative idea, I have met with many cases in which it appeared to be a steady morbid growth which had been for long under the surface, but through some accidental cause had been brought into sight. Shock may produce the effect. It may be one sudden shock, a repetition of shocks, or the result of a prolonged state of expectancy and anxiety; thus a person who has been in imminent danger for some time may become so affected that the dread is never overcome; allied to this I have met with instances where a dream of unusually vivid character has been enough to start the whole train of morbid ideas and has fixed them for ever. There is no doubt that the nervous system under certain conditions is more prone to take these impressions and to retain them. It seems as if through certain surrounding conditions an impression is made which reaches the more fixed and automatic part of the nervous system so that it is no longer merely stored for use when it is called for by consciousness, but it appears as a reflex act, one impression bringing it up without any will being exercised. I believe that these imperative ideas are very common and that nearly everyone has some. One man cannot dissociate from his mind some notions which have no apparent relationship; these may, very probably, have arisen in the pre-conscious stage or in the stage of development before memory became established. I have the feeling, which is common I believe, about walking along a pavement. I have an inclination to avoid the cracks and at the same time I have a tendency, I own, not irresistible, to touch the iron railings with my stick when I walk along a street, but more still, I hardly ever go to sleep without just before becoming unconscious having one sentence which has relation to nothing in my past or present, but which forces itself on my mind, then I know I am going off to sleep and if for a time I have been sleepless and this sentence comes to me I know sleep is not far off.

To compare mind and body we know that what is one man's food is another man's poison, that a very trivial thing apparently will make all the difference, thus the comfort of one depends on his not taking tea, while another cannot take strawberries. The body has its imperative ideas, and I shall have to refer to the imperative ideas of the senses or rather the imperative sensations—one person being unable to stand the presence of a cat, while another is affected by a rose. The senses, in fact, give us valuable aid in considering this question, for not only can we see that these imperative nervous influences may be primary, as with the cat smell, but they may be secondary or associated; thus in the so-called photisms certain persons have associations with a sensation, which have no real relationship to them. For example, a man hears a vowel sound and sees a certain colour at the same time, whereas another perceiving a certain smell also sees a certain colour arise with the olfactory sensation. These sense relations are very imperative and are so early established that most of those who have them cannot go back to any moment when they were free from them.

In most books on the subject of recent date it will be seen that there is shown to be a very near relationship between these imperative ideas and the neurasthenic condition, also that neurasthenia depends to a great extent, if not entirely, on faulty association of ideas. The process has thus been described: "In ordinary course of ideation acting along ordinary lines of association, one or more ideas insert themselves and remain in consciousness with morbid intensity and persistence." Such ideas differ from delusions, hallucinations and illusions from the fact that the patient recognises that they are false. Patients may have hallucinations and illusions and not believe them, but these have a different relationship to the mind from the persistent sensations of the patient with the imperative disorder, the hallucinations are less regular in their recurrence and are not as a rule started directly by some external sensory impression. The imperative ideas vary in degree. In certain conditions a word may always insert itself, or a word may seem to escape when wanted, leaving a miserable feeling of a void which prevents rest; this is in one way like the insanity of *doubt*, to which we must refer later. Beside the simple insertion or escape of words, the word may bring up always an unpleasant association; thus the idea of dirt, of blood, of impurity, moral or religious, may arise, or certain melodies may spring up and recur without any recognised excitant. There are other allied forms of disorder in which there is not only the morbid association, but also a tendency to act on the idea. They are what a patient in Bethlem, who was very impulsive and dangerous, called a "suggestive consciousness." An idea came into his mind and had to be acted on at once. In his case there was an uncontrolled mental reflex; idea suggested action without any regard to consequences. Other examples are those who are affected by heights, by rapidly passing trains and the like. I believe nearly all such cases belong to highly neurotic families. I have been struck with the extreme horror of heights and movements which is present in those whom I have known to be neurotic. Some of these persons suffer from the dread that they shall act upon their ideas; thus one will dread a razor or a rope, while another will avoid children, lest she should push them over a cliff. In other cases we get more organised notions, and terms have been coined to express them; thus the persons who dread solitude and those who avoid multitudes have been described as suffering from claustrophobia and agoraphobia respectively. I have recently seen a clergyman who for years has been obliged to get his wife to walk about a yard behind him, because he had an unconquerable feeling that someone would jump upon his back. Doubtless there are very many other special forms which hardly deserve to have names, so long as their origin is recognised. The most definitely and elaborately organised state of this kind is seen in many cases suffering from the so-called paranoia.

The imperative ideas may arise chiefly with the senses. I have seen several cases in which the idea that there were pins or needles in the clothes rendered the patient's life miserable. In one case a young lady with neurotic heredity asked over and over again whether it were better for her to put on her clothes, still having doubt as to the presence of pins, without examining them, or if she had better spend half-an-hour searching in vain for the pins, and then still not satisfied, put on her clothes. I advised the putting them on without consideration, and whether this or other treatment had the effect I cannot say, but any way she recovered completely. The persistent feeling of dirt or of communicating contagion are allied states to those just considered; they differ only in the explanation. In one case a middle-aged maiden lady spent all her time in dusting her home treasures, still being wretched at their supposed state of dust. In other cases a feeling that the contact with the door handle may either give or communicate poison is not very uncommon; in many of these cases there is some sexual vice, or at least some sexual abnormality. One spinster was miserable for hours after she had passed water or touched the chamber utensil; she was in the habit of putting on gloves to prevent harm arising. In another case the patient used to wear gloves lest her hands should touch the genitalia. Other notions equally imperative and equally unreasonable abound. In one case a young man otherwise healthy but of neurotic heredity was haunted by the fear that fire was lurking in all sorts of places; he thought when in my consulting room that fire might exist among the books on the table and asked to be allowed to see. He opened the pages of the various books and for the time was at peace only once more to wish to see that he had not by friction set up fire in the books he touched.

I have already said that some of the dominating ideas arise from habit, and I suppose all of us have experienced the inability to be satisfied that the front door was locked or the gas turned off. In some cases a hallucination may become an imperative idea, and in one very interesting case the hallucination had formed what I may call a mental scar. A woman had an attack of puerperal melancholia, and in this she saw her brother's throat bleeding and cut, and she was convinced that her brother had cut his throat in grief at her madness. No evidence could satisfy her that her brother was well. After months of illness she got quite well, but this hallucination reappeared as a kind of warning when she from nursing or the like was overwrought. There seemed to be a denudation exposing the established scar. Akin to these imperative ideas connected with sensation one must not forget certain hypochondriacal cases in whom there is but one set of morbid ideas, which, however, dominate and destroy the usefulness of life. There are several medical men in London who have such imperative ideas; they believe they are dying, that their brains are unlike those of other human beings or that their general nervous system is one apart. No argument will satisfy these persons. Therefore, in a certain way they differ from very many who will admit that their ideas are unreasonable though they are uncontrollable. As a rule it is more common to meet with imperative association of ideas; these may be linked in many ways, but one idea brings up as a certainty the corresponding one.

Years ago I had under my care a man who, having lived in India for some years, was once with others in great danger from a rising of the natives. It was hourly expected that their fort would be stormed, that the men would be killed and the women outraged. So it was agreed that the men should, if occasion arose, kill their womenkind and then fight to the death. They were relieved, but the association between wife and killing was so firmly established that the patient felt his only safety rested in his living in an asylum. This he did till his wife died; after that he went out and lived at liberty.

Volition is affected in several ways, but the chief disorder is shown in some of the cases of *doubt*, in which the patient is never able to make up his mind as to what to do. In some there is the doubt about movement. I have letters in which patients ask over and over again if there is any harm in their going more and more slowly, if by so doing they can satisfy themselves.

I have known a young man of this type who nearly lost his life as a result of not being able to make up his mind; in the particular instance it was a question whether he should shoot at a bear, and if a companion had not, his future chance would have been done for.

Among the associated ideas which are imperative some are strongly allied to the emotional side. One has met patients who seemed to have had their emotions perverted so that they invariably laughed when they ought to have shown sympathy. In some cases the sexual attraction which may be either normal or abnormal may be persistent, recurring and dominant, and there are certain associations of this kind specially noteworthy. Thus certain associations produce ideas of sexual impotence, or some very slight things may during the earlier days of marriage set up imperative ideas which produce loathing, hatred and impotence, which will be dangerous to one or both of the parties. A young married man finds from certain acts that his wife is not a virgin, or he may simply get the notion into his head and it may dominate his life.

Similarly the judgment may be in abeyance as in the "*folie du doute.*" I do not for a moment intend to assume that the parts of mind are distinct entities, but it is well to note that certain faculties are more affected in one case than in another. In some cases there is a morbidly scientific spirit. One patient who was under me in Bethlem could not resist the temptation to see whatever was written if he could, so that in a train or an omnibus, in a house or in a dining room, if a man opened a letter he could not help trying to read over the owner's shoulder. He was struck by the owners of the letters several times, but still he felt the overmastering desire to know. He is *Grubelsuchtic.* He has several times been in danger of his life when, seeing a piece of paper revolving round a cab wheel, he has tried to get it. He has gone home miserable after failing, and has returned to the street at night to see if he could find it. This man has been for the last twelve years in this state and from time to time comes to see me, for I have been anxious to watch his course. He is no more weak-minded than he was and manages to earn a living, though his peculiarity interferes with his doing full work. As to the curability of these imperative ideas, I can only say that if they have existed for a year or more I do not think there is any prospect of cure. If they are acute and if associated with any special cause, or if associated with a period of life, such as adolescence or the menopause, there is some slight hope. Few of these cases need to be permanent inhabitants of asylums, in fact, many of them need never be there if their friends can afford to keep them out of asylums.

Then, to conclude, in my experience there occur certain organic nerve defects associated with abnormal mental reaction which may show themselves on the organic or visceral side, on the sensory side, either simply or by association; on the voluntary side with morbid impulse, and on the intellectual side by a further development of the association of ideas, these latter being more commonly called imperative ideas, though they do not differ from the sensory or visceral disorders.

3.—Dr. C. Mercier.

The feature in Dr. Tuke's paper which impresses me most, is the complete acceptance of a doctrine that I have long held and preached, but which has never before, as far as I know, had any adherent, that insanity and disorder of mind are not convertible terms, and that disorder of mind—grave and prominent disorder of mind—may exist in a sane person, and without the question of insanity being even raised. Another favourite doctrine of mine, on which I am fully in accord with Dr. Tuke, is the importance of noting the earliest departures from the normal if we would discover the true nature and origin of mental disorder. It is in this connection that the observations of Dr. Wilks are so interesting. That the trifling forms of obsession to which we are all liable—the persistent and often annoying recurrence in memory of a word, a line of poetry, or a few bars of an air—are of the same nature as the graver imperative ideas of which Dr. Tuke has given us so many instances, there can, I think, be no doubt, and hence it becomes important to note the conditions under which these trifling forms occur.

Speaking from my own experience, they do not occur when the mind is fully occupied and the attention engrossed in some intellectual pursuit—in reading, writing, calculating, playing chess or whist. Nor do they occur when the body is inactive and the mind is pursuing some train of thought—say on a railway journey—nor when bodily activity is accompanied by close exercise of attention, as in games of billiards, bowls, &c. The conditions under which they occur, and under which they seldom fail to occur, are when we are undertaking some bodily labour of a more or less monotonous character, which demands considerable muscular activity, which does not require close or unremitting attention, but which calls for so much and such frequent exercise of attention as prevents us from carrying on any continuous and connected train of thought apart from that devoted to what we are occupied about. It is, in short, when we are engaged in some handicraft, such as carpentering, joinery, smith's or other metal work, gardening, and such like occupations, that these strains of reminiscence arise and haunt us, and become, for the time being, imperative ideas. If, then, we can identify the psychological condition that obtains during our occupation in these handicrafts, we can get some insight into that which permits of the occurrence of obsession in its graver forms.

In the graver cases of the malady we must suppose that a local portion of the brain has attained a habit of spontaneous activity which overpowers the control of those higher regions to which by its anatomical position it should be subject—that at length the whole concentrated and focussed energy of the higher nerve regions, the local direction of which is the physiological basis of will, is insufficient to overpower the rebellious activity of this revolting section; whether the imperative idea remains an idea, or whether it finds expression in word or act, is purely a matter of degree, and affects in no wise the explanation of the physiological process which is the basis of both idea and act.

The psychological condition in the slighter forms of which mention has been made appears to be this: there is a considerable degree of alertness of attention, and at the same time the actual pabulum that is supplied to the attention is insufficient to keep it fully occupied. The attention is concentrated upon the work in hand, not continuously, but at intervals only; and in the intervals, during which it is not so occupied, but is still alert, it has presented to it pabulum from the field of sub-consciousness, about which it becomes occupied; and the states which should normally be subconscious are thus lifted into the light of full
ciousness, and become subjects of attention without direct
ce from the will, which is otherwise engaged. A state
has once thus arisen and presented itself to the attention
unoccupied interval, tends, so long as the work is approxi-
ly the same, to repeat itself in obedience to the law of association, and thus it happens that the image, the musical air, the form of words, which has once arisen in one of these unoccupied intervals of attention, tends to occur again and again as these intervals are repeated. Briefly, the psychological condition may be stated as the capture of the attention during the abeyance of the will. If we take this expression and apply it to the more serious of the cases that have been brought forward, we shall find that it applies to them also. The imperative idea is in every case one that forces itself—if I may be allowed for a moment to speak metaphysically—upon the attention, and that cannot be, or at any rate is not, excluded by the will. The will is, so far as that subject of attention is concerned, in abeyance.

The physiological process which underlies this psychological condition appears to be this: returning now to the trifling and quasi normal instances of the malady, those instances in which an idea repeats itself with undue frequency and force during the execution of some handicraft operation. The operations of the handicraft necessitate and imply great activity of the middle centres. At the same time there is very little call for the activity of the higher centres. No general, or intense, or widespread activity of the centres which are the substrata of elaborate, intellectual operations, and especially of verbal operations, is required, nor is there in the work in hand any outlet for such activity if it is evoked. Yet the high activity of the middle centres cannot go on without spreading, to some extent, in an upward direction, and starting activity in the more unstable and more easily discharged centres above them. It cannot, at least, fail to evoke some activity of these higher centres in those persons in whom activity of these higher centres is habitually great, and their tendency to have this activity easily evoked is consequently also great. During the handicraft operations, certain of these higher centres are therefore started, and in the case supposed they are centres the substrata of verbal utterance. Hence forms of words—successions of words—arise in the mind. Were these higher centres the seat of widespread activity, proceeding normally, its focus swaying hither and thither as multiform successions of verbal utterance arose, the activity started by the spread of discharge from the middle centres would in great part be drafted off to the scene of greatest activity, and what ideas accompanied the remainder would be overpowered by the more vivid and voluminous ideas thus rendered possible, would remain subconscious, and would never occupy the attention. But since these small areas, started by the upward spread of the discharge, are practically the only scenes of localised activity in the higher centres, the ideas which they represent are practically the only verbal ideas present in consciousness; and as such they seize upon the attention in every vacant interval, and are thus brought prominently to the front. That local concentration of widespread energy which is the physiological equivalen of the will, is directed upon the middle layers and goes to direc the handicraft operation. It passes by the active articulatory centres and leaves this latter unenveloped in its rushing volum free to assert itself, to set up a somewhat independent and alm isolated centre of discharge, which has its mental equivalen the imperative idea.

4.—Dr. J. Milne Bramwell.

At the conclusion of his interesting article on "Imperative Ideas," Dr. Hack Tuke states that the mental phenomena which he has described have been more clearly recognised by French and German, than by English writers. This is undoubtedly correct; but, although the former have long recognised such conditions, it is only within comparatively recent times that they, like ourselves, have ceased to confound them with various forms of insanity. Dr. Ladame, of Geneva, for example, in referring to the different opinions expressed in reference to "folie du doute" and "délire du toucher" by Schüle, Magnan, Krafft-Ebing, Marcé, Jules Falret, Morel, Lasègue, Ball, Meynert, Kræpelin and Scholz, says "folie du doute" is regarded by contemporary writers sometimes as a symptom of the most varied mental affections, sometimes as a psychopathic episode of hereditary degeneracy, sometimes as a special form of psychosis, and sometimes as a simple elementary psychic trouble, dependent upon the general pathology of mental alienation. Thus, doubt, he says, does not only exist among the patients, it has passed into science and could equally be called "folie du doute" on account of its uncertain place in the chart of mental maladies, as well as for the strange symptoms which characterise it.

According to Ladame, Pinel at the beginning of this century first commenced to recognise "folie du doute," while Esquirol in 1838 published the first detailed clinical observation of "délire du toucher" and laid stress on the patient's constant fight against obsessions and his recognition of their absurdity. Brierre de Boismont in 1853 also stated that the irresistible idea keenly combated by the patient was characteristic of these cases. "There is a distinction to make," he said, "namely, that ideas may make one ill when they dominate the mind, but one is not eally mad except when the will has become powerless to control e impulsions." That the patients fight against their obsessions ithout succeeding in getting rid of them, was particularly insisted pon in the discussion upon monomanias at the Medico-Psychogical Society of Paris on June 26, 1854. Delasiauve in 1859 ade a first attempt to distinguish between pseudo-monomanias d recognised forms of insanity. After 1860 observations on élire du toucher" became more numerous, and Baillarger pears to have been the first to notice that these conditions frequently commenced at puberty. Marcé in 1862, though he failed to separate "délire du toucher" from insanity, clearly described the origin of imperative ideas. "In a predisposed person," he says, "feeble of character, endowed with keen sensibility, a word, an emotion, a fear, a desire leaves one day a profound impression. The thought, born in this manner, presents itself to the mind in an importunate way, takes possession of it, does not leave it, dominates all its conceptions; during this time the individual may have consciousness of all the absurdity, unreasonableness or criminality of this idea, the acts themselves soon conform to these unhealthy preoccupations and become absurd and extravagant." The term "folie du doute" occurred for the first time in the celebrated discussion upon "la manie raisonnante" at the Medico-Psychological Society of Paris, March 26, 1866, but from what was said by Jules Falret on that occasion it was evident that what he called "folie du doute" corresponded to "délire du toucher" and not to the form of mental trouble described two years later by Griesinger as "Grubelsucht" and "Krankhafte Fragesucht." In this discussion Baillarger stated that one of his patients never ceased to make suppositions and to lose herself in "if" and "perhaps." A clear distinction between these diseases and recognised forms of insanity was made for the first time in 1886 by Morel, and he stated that patients suffering from imperative ideas did not interpret their obsessions after the manner of the insane, that they neither experienced hallucinations nor illusions, nor underwent those transformations which change the personality of the insane and make them radically different from what they were before. Heredity, not only hereditary insanity, but also other nervous conditions such as hysteria and hypochondria he regarded as important exciting causes. In 1868 Griesinger published cases which showed for the first time that the entire malady might consist in insoluable questions, which unceasingly pursued the patient who could not escape from "why," and "how," and he considered the condition might be independent of emotional complications. In 1875 Legrand d Saulle showed that the same patients could present successive the symptoms of "folie du doute" and those of "délire toucher," and he attempted, by joining these two conditions, form a special mental affection having a veritable morbid entit In 1877 Westphal published an important contribution to t subject. According to him, the obsession never becomes a t "idée fixe délirante," but always remains a stranger to patient's ego, while the insane conform logically to the deductio of their fixed ideas. This scientific distinction between fi ideas and obsessions has long been recognised by the Chur which has always made a difference between possession and obsession, saying for example: "This man is not possessed, he is only suffering from obsession." According to Westphal an obsession is not an emotion, nor is it ever produced by one, and if attacks of mental agony appear later, these are always secondary and simply concomitant phenomena. This opinion is opposed to the observations of Morel and the French savants. Wille holds that obsessions can have an emotional base, and thinks that they are sometimes followed by veritable mental alienation, while Westphal affirms that cases of obsession never become insane. Wille, as well as Legrand du Saulle, has noticed the frequent suicidal ideas and even attempts at suicide, sometimes followed by death, and considers "folie du doute" and its likes as an intermediate condition between the nevroses and the psychoses, and that obsession is always ready to pass into madness.

Westphal divides obsessions into three kinds:—(1) Those which remain purely theoretical as the "folie du doute," when it takes the form of questions. (2) Those which produce certain actions as the "délire du toucher." (3) Impulsive obsessions which provoke immediate actions.

While other authors since Morel have emphasised heredity as an essential factor in obsessions, Magnan in 1885 appears to have been the first to consider this mental trouble as a direct and immediate sign of morbid heredity.

In opposition to Legrand du Saulle, Ladame demands the separation of "folie du doute" and "délire du toucher" into two distinct clinical varieties. These, like pleuro-pneumonia, are certainly often associated, he says, but more often exist isolated. In illustration of this he cites the following cases:—

(1) A young man, aged 28, suffered from fear of contamination and frequently washed his hands as a method of purification. He never feared he would forget anything and never addressed uestions to himself. This, according to Ladame, was a case of délire du toucher" without "folie du doute."

(2) Miss ——, aged 33, has since childhood had ideas which
could not get rid of. She asks herself all sorts of questions and
ks in vain for their answers. These are particularly in refer-
e to the other world and the creation. Did everything make
lf? Has God created all things? Did not the world make its-
? Is there a God? How can one divide objects into infinitely
le parts, when each little part can still be divided? How is it
t an object infinitely divided can still be divided, notwithstand-
that one cannot divide it any more? Is God able to divide it

still further? God only can divide it and nevertheless this particle cannot be divided! How do you account for that? She has never suffered from "délire du toucher."

Van Eeden says that in four cases treated by him the connection between "folie du doute" and "délire du toucher" could not be mistaken, and that in all these cases the "folie du doute" was evolved after the "délire du toucher" and was an evident consequence of it. One case commenced with the fear of catching syphilis. The patient avoided touching anything he suspected of being contaminated and, if this happened, scrupulously washed his hands. His precautions became exaggerated, until he not only doubted their efficacy, but also the clearness of his judgment, and then, despite the excessive attention given to these washings, could not obtain a certainty of complete cleanliness. A similar progressive evolution was observed, says van Eeden, in another case, where the patient suffered from "crainte de souillure," then doubt and finally mistrust of herself. The "délire du toucher" began with fear of soiling her food, she washed her hands incessantly, but afterwards the doubt returned. Another patient, who was nursing a case of cancer, became afraid of catching it. She washed her hands constantly and avoided coming in contact with suspected persons.

In agoraphobia, claustrophobia, &c., van Eeden says the idea of fright arises suddenly from the impression of the surroundings, and it is only after having this that the patient avoids open or closed spaces. Here it is the fear which causes the obsession. He classifies these and similar conditions as follows:—(1) "Conceptions obsédantes"; here the obsession springs from a precise and detailed conception of some act which acquires an impulsive force. (2) "Les émotions obsédantes"; chiefly terror, in which a momentary emotion, an impression—which a normal man could equally well experience, but which he quickly represses—dominates the volition and the reason. The patients are not able to sa what they fear, or if they give a reason it is evidently an invente one. (3) "Impulsions obsédantes"; the irresistible tendency commit strange or improper acts. (4) "Les idées obsédante properly so-called; the intellectual obsessions of the French, t "Grubelsucht" of Griesinger and Berger. The patient is not a to escape from the obsession to think constantly about a ce subject or question. Here it is neither a question of a perc or emotion, nor of an impulse to commit an act; there is one isolated idea—a word, a phrase, incessantly pursue patient and continually occupies his thoughts.

Van Eeden regards what he calls "manias of superstition" as an interesting variety of obsessions. One of his patients, a man aged 40, of healthy constitution, has since childhood attached prophetic signification to puerile facts and events. To wear a certain necktie promises him happiness or unhappiness. If he does not touch a certain boundary he thinks evil will happen to him. If he does not re-read a certain line or make a certain letter thicker in writing something horrible will befall him. At first his strange ideas were insignificant, or he was able to resist them, but as he grew older they filled his life and rendered it intolerable. For twenty years he made a pilgrimage every Sunday to the railway station in order to kick a certain post three times with each foot. If he did not do this his father would die. In order to rid himself of these obsessions he makes vows and associates threats with them. He says for example: "If I yield to one of my caprices in the course of an hour I shall have apoplexy before twenty-four hours have past." At first this succeeded, but soon the effect of the vows diminished, and he was compelled to make them stronger. The unhappy man now stands sometimes for a quarter of an hour muttering the most fearful imprecations, in order to get the strength to go an errand. If he omits them he is forced to obey the most absurd impulses. He must stop before a certain house, retrace his steps, touch boundaries, stop passers-by or touch their clothes; in a word he is obliged to act like a maniac. His intellect is perfectly normal, and he attends to his business as if nothing were the matter.

Bérillon thinks the professional character of these nervous troubles has not been sufficiently noticed, and he draws an analogy between them and the different functional spasms which show a tendency to professional localisation. In illustration of this he cites the following cases:—A young priest, not timid in the performance of his other religious duties, suffered agony on entering the pulpit. Another suffered in the same way when he received a confession. A medical student suffered xtreme agony at the sight of a few drops of blood. A chemist ade up a prescription which caused the death of a customer. e was able to prove that it was dispensed exactly as ordered by e doctor, but as his existence became a veritable torture from nstant fear of making a mistake he sold his business. A notary ly had morbid fears when he had to give a professional opinion. hairdresser noticed that his hand trembled one day, and then stantly dreaded that this would reappear when he shaved his st customers. The same anxiety did not exist when he had to shave a poor or unknown customer. Dr. Frémineau reports the case of an actor who abandoned his profession on account of extreme stage fright. This condition only appeared after a successful career. Dr. Bérillon reports several similar cases. Riegler has noticed a morbid fear amongst railway mechanics, to which he has given the name of "sidérodromophobie"; this is characterised by an extraordinary aversion to their habitual occupation, and the sight of a train or the whistle of an engine is sufficient to revive their anxiety. Grasset mentions that a distinguished Parisian surgeon commences to be anxious the moment a patient leaves his consulting room with a prescription. He anxiously asks himself whether he could have written centigrammes instead of milligrammes, and only recovers his mental calm when his servant, sent to seek the patient, brings back the prescription and he can see that it is all right. Another doctor, he says, is rendered perfectly miserable by the fear of microbes. Brochin reports the case of a doctor who fears no contagious malady, except diphtheria, and who shows proof of veritable heroism every time he sees a diphtheritic patient. A case has recently been reported from abroad, where a medical man, dreading that his fees might be the means of contagion, invented elaborate methods of sterilising them, and I know of a similar case in this country.

These morbid fears are awkward enough when they occur in ordinary life, but according to Bérillon they acquire graver importance when the subjects of them are liable to enforced military service, especially in the ranks. In Legrand du Saulle's classic case of agoraphobia, a lieutenant of infantry experienced undefinable agony when obliged to cross an open space in civilian clothes, but this never happened when in uniform. When an officer suffers in this way he can escape from the intolerable situation by getting leave of absence, but it is not so with the soldier. Bérillon gives amongst others the following examples:—

Paul C., aged 25, illness dates from military service. When ordered to cross the horizontal bar at the gymnasium he was seized with extreme apprehension, and afterwards the idea of thi exercise was always present. Again ordered to perform it h became terrified, and asked to be excused, but from this time hi superiors insisted more and more upon the dreaded exercise One day in attempting to cross the bar he became giddy and fe upon his head. A veritable agoraphobia developed, and since h return from military service he has been unable to cross a road himself.

M., aged 37, commercial traveller, suffered from chronic diarrhœa, which caused him to dread leaving home; after this ceased he retained his nervous fears. Once, when a short distance from his house, the anxiety was so strong that he was obliged to return, and since then, seven years ago, he has lived in a circle having a diameter of about 200 metres, and nothing will induce him to leave it. When, as a member of the Reserve, he had to serve his first period of twenty-eight days he was able to perform his duties, but at the second period the agoraphobia had developed, and by dint of diplomacy, ingenuity and bribery, he managed to be kept constantly employed in the barracks.

According to Dr. Gélineau a crowd of sentiments of repugnance, &c., which the laity group as aversions closely resemble the conditions we are discussing. Henry the Third, for example, who showed his bravery at the Siege of La Rochelle and elsewhere, could not bear the sight of a cat. The Duke of Epernon fainted at the sight of a young donkey. Ladislas, King of Poland, got frightened, and ran away when he saw apples, and Favoriti, a modern Italian poet, could not bear the smell of a rose. Dr. Pierre d'Apono was so frightened at the sight of milk and cheese that he fainted. Montaigne says: "I have seen more people driven to flight by the smell of apples than by arquebuses, others frightened at a mouse, made sick by the sight of cream, or by seeing a feather bed shaken."

Ribot applies the term "fixed ideas" to the states we are discussing, and regards them as "chronic hypertrophy of the attention"; the fixed ideas being the *absolute*, attention the *temporary* predominance of an intellectual state or group of states. The fixed idea is attention in its highest degree and marks the extreme limit of its power of inhibition. There exists, he says, both in normal attention and in fixed ideas, predominance nd intensity of a state of consciousness; this is more marked owever in the fixed idea, which is permanent and disposes of e important psychical factor—time. "In attention this excep- nal state does not last long; consciousness reverts spontaneously its normal condition, which is a struggle for existence between erogeneous states. The fixed idea prevents all diffusion. re is no antagonistic state that is able to overthrow it. Effort mpossible or vain. Hence the agony of the patient who is scious of his own impotency." The following is Ribot's con- ion of the probable physiological condition associated with ideas:—"In its normal state the entire brain works; diffused ity is the rule. Discharges take place from one group of cells into another, which is the objective equivalent of the perpetual alterations of consciousness. In the morbid state only a few nervous elements are active, or, at least, their state of tension is not transmitted to other groups. Whatever may be their position in the cerebral organ, they are as a matter of fact isolated; all disposable energy has been accumulated in them, and they do not communicate it to other groups; whence their supreme dominance and exaggerated activity. There is a lack of physiological equilibrium, due probably to the state of nutrition of the cerebral centres." Ribot refers to Westphal's recognition of the difference between fixed ideas and insanity, and his statement that "the fixed idea is a formal alteration of the process of ideation, but not of its content." The "formal" perturbation consists, says Ribot, in the inexorable necessity that compels the association always to follow the same path. "There is alteration, not in the nature, the quality of the idea, which is normal, but in its quantity, intensity and degree." Thus it is perfectly rational to reflect upon the usefulness of bank notes, or the origin of things, and this state differs widely from that of the beggar who thinks himself a millionaire or the man who believes himself to be a woman.

Treatment.

The following cases, from my own practice and that of others, illustrate the value of hypnotic suggestion in cases of "imperative ideas."

(1) Mr. A., aged 24, consulted me in May, 1889. Some months previously he had a number of diseased glands removed from the face and neck, and went up the Mediterranean to recruit. While crossing a plank he fell and injured his perineum; an abscess formed, which burst externally and into the urethra. When I saw him there was a large unhealthy wound, through which th urine escaped. I instructed him to pass a soft catheter regularl and the wound became more healthy. One day he was impelle to empty his bladder before he could pass the instrument and t water again escaped from the wound. This happened more a more frequently; at last the idea of passing water at once caus him to empty his bladder, no matter where he was at the ti This appeared to be entirely independent of the physical condit of the bladder, which did not contract because it was full or comfortable, but because the idea of urination presented itsel the patient's mind and was instantly translated into its phy equivalent. He now thought constantly about his condi

which became greatly aggravated in consequence. He began to sleep badly and awoke frequently during the night; the instant he did so he thought of his bladder and was immediately compelled to empty it. Despite all treatment this continued for several months, and his state became a grave one. I had not previously employed hypnotism, but the mental element in this case seemed so marked that I determined, as other treatment had failed, to try what it would do. After explaining to my patient, an educated man, that I had no practical, and only slight theoretical, knowledge of the subject, I proceeded to hypnotise him by Braid's method. In a few minutes his eyeballs rolled upwards and inwards and he became lethargic. I repeated this the two following days, and then suggested during hypnosis that he should cease to think about his bladder, should always be able to pass his catheter, retain his urine eight hours and sleep well. These suggestions were immediately fulfilled; from that day there was no return of his troublesome symptoms and the wound healed without operation in about twelve months.

(2) Mr. B., aged 25, first consulted me March, 1890. Formerly strong and athletic, distinguished football player, bicyclist, &c. Two years previously, after the death of his mother from cancer of the breast, he began to fear that he might contract the same disease. This idea grew stronger and stronger, he developed neurasthenic symptoms, and suffered from insomnia, depression, dyspepsia, &c. Finally, the dread of cancer passed into the firm conviction that his left breast was affected by it. He now remained nearly always in one room and would not pass into another without muffling himself up and putting on an overcoat. For some months he complained of difficulty in moving the left arm and carried it in a sling. I found nothing to justify his fears, but the muscles of the arm were distinctly wasted from disuse. He was easily hypnotised at the first attempt, and this was epeated nearly every day for a fortnight, when deep hypnosis vith somnambulism was obtained. His morbid ideas at once disppeared and his general health speedily improved. A few days ter the treatment was abandoned, I saw him driving a spirited rse and holding the reins in his left hand. A week afterwards told me that he felt perfectly well, and was just going to jump oung horse over some hurdles. Up to the present date there been no relapse.

(3) Mr. C., aged 30, consulted me July, 1890. Had good lth up to six months ago, when he was frightened by being kened by his sister, who had acute mania, entering his bed-

room. From this date he suffered from insomnia and dyspepsia, lost weight and strength, became melancholic, shunned society and had the fixed idea that he would become insane. Hypnotised five times from July 3 to July 7, 1890. Deep hypnosis was easily induced at the first attempt and he slept well each night. Recovered. No relapse up to May, 1891, when he died of influenza.

(4) Mr. D., aged 43, first consulted me September, 1892. His illness commenced in early childhood, with the fixed idea that everyone noticed and criticised him and he soon began to blush if looked at. This condition had continued ever since and had latterly become worse and made his life a torture to him. Not only did he blush in the most excessive manner when anyone, even his own children, looked at him, but the thought that anyone might look at him was sufficient to make him blush even when alone. He felt that he must give up business and had suicidal ideas. He suffered from various neurasthenic symptoms. I attempted to hypnotise him five times without result. He returned for treatment in April, 1893; his condition was worse and he was in despair about himself. I hypnotised him forty-two times up to October, 1893. Slight hypnosis was induced at the fifteenth attempt, and afterwards became fairly deep, when he commenced to improve and before the close of the treatment his morbid symptoms had disappeared. Recent news confirms his recovery.

(5) Mrs. E., aged 36. Educated and highly intelligent. One of her brothers is congenitally deficient in intellect, and she has suffered from fits of depression since childhood, which have become more frequent and prolonged during the last fourteen years. The mental condition of one of her children, born during one of these periods, causes grave anxiety, and a younger child stammers. Four and a half years ago, after the death of a relative who had exercised an unfriendly influence on her life, she constantly thought that various misfortunes that were happening t
her would give him pleasure; this idea increased and develope
until she felt that he knew everything she did and gloated ov
her troubles. Finally she became possessed of the idea that s
was haunted by him and felt conscious of his presence. H
whole life was filled with these thoughts, and she ceased to c
for society or intellectual pursuits. She was constantly depress
slept badly, and was weak physically. I first tried to hypno
her on November 26, 1892, and repeated the attempt a hund
times without success, until April 25, 1893; her condition me

ile growing steadily worse. Slight hypnosis was induced on
il 26, and repeated twenty-three times up to July 21, 1893.
ediately after the first induction of hypnosis she rapidly
oved and the morbid ideas and depression entirely dis-
red. She quickly regained strength and interest in life and
he present date there has been no relapse.

Mrs. F. I have received the following notes of this case
. F., the patient's husband: "My wife has suffered from
ma, following influenza; she had low temperature, loss of
ness of intellect, slowness of movement, general irregular
aof the body, facial disfigurement, alteration of voice and
af pains. I put her on thyroid extract in January, 1893,
hcugh the symptoms peculiar to myxœdema disappeared,
hene utterly sleepless, her limbs trembled after the least
a ynd her digestion remained very bad. I brought her to
haarch 1, 1894, to see whether suggestion would procure
the second attempt you succeeded in inducing very
heanosis and she began to sleep fairly well. For more
awar she had never had more than three hours' broken
d often far less. She soon began to sleep thoroughly well and uninterruptedly, and her digestion, for which I had found drugs and careful dieting ineffective, disappeared after a few suggestions. Her legs became stronger and her energies restored very much to what they were twenty years ago, when she was renowned amongst her acquaintances for her untiring activity. But the fact that strikes me most forcibly is this. Several members of her family are sleep-walkers. She used to walk in her sleep in childhood, and once or twice as a young woman, and the habit is transmitted to my youngest girl. When her first baby was born—sixteen years ago—the thought crossed my wife's mind: 'What if I walk in my sleep and do an injury to my child?' I endeavoured to persuade her that she had grown out of
he habit, but the attempt was wholly fruitless. The idea grew
ntil it assumed the character of an 'idée fixe' and she always
ed herself to the bedpost at night. All attempts to break her-
lf of the habit were failures, and if she went to bed without
tening herself she was never able to go to sleep until she did so.
hen we moved into our present house, three and a half years
, she became alarmed at the great height of the bedroom
ndows from the ground and their lowness from the floor. She
an to suggest that possibly she might undo her own knots
ng her sleep and get out of the window. I pointed out how
kely it was that she should walk in her sleep after a score of

years complete immunity. She granted my reasoning was just, but it did not dispel her fear, and she insisted upon my tying her to the bedpost each night in a very effective manner. In May, 1894, I told you of this persistent dread and asked you to suggest that she should neither walk in her sleep, nor be apprehensive of so doing. During that *séance* you repeated this suggestion two or three times, but have not done so since. The effect was magical. She has never asked me to tie her to her bed from that day and tells me she has never once thought about it. To me it is all the more remarkable as the hypnosis in her case is so slight and appears to pass into natural sleep if you leave her for a few seconds. She is of a nervous, excitable temperament, but by no means greedy of the marvellous or ready to accord belief to any doctrine. She had a healthy scepticism of the possibility of any-one hypnotising her, but was anxious for the attempt to be made as she suffered so acutely."

I saw this patient twelve months later, when she told me she remained entirely free from her morbid fears.

(7) Mr. G., aged 28, first consulted me April, 1894. His father was very nervous and passionate and had suffered from "brain fever" and chorea. The patient is slight and undersized, and suffers from various neurasthenic symptoms. At the age of 14, he had many religious doubts and fears, and believed he had committed the unpardonable sin. At the age of 16, while working in a cocoa manufactory, he began to fear that the red lead which was used in fastening certain hot pipes, might get into the tins containing cocoa and so poison people. This was the commencement of a "folie du doute" and "délire du toucher" which have never since left him. Instead of going on with his work he was irresistibly impelled to clean and reclean the tins. The following is taken from the letter of a friend to whom he confided his troubles: "On October 1, 1891, he told me that he ha attempted to commit suicide, as his life was so miserable [he ha taken poison]. He had read of a case of poisoning through ea ing chocolate and connected himself with it, though it was fi years since he had helped to manufacture any. He now believ he might have been careless with the moulds and thus have p duced a poisoned chocolate, which years afterwards had cau the child's death! The grotesque absurdity of the story, as related it to me, would have made me laugh, had I not felt h terribly real it was to him. His vivid imagination had pict every incident of the tragedy; the child buying the choco running home full of happiness, then becoming ill and grad

sickening in awful agony till released by death. The keenness of mind with which he sought to prove the reasonableness of his belief that he had poisoned the child was extraordinary. He wrote: 'Yesterday I was unscrewing some gas burners in a provision shop and got some white lead on my hands, and I have been thinking that it may have got amongst the food.' I found that brooding over this fancy had brought him to the verge of despair and for weeks his life was a perpetual agony. He has worried himself about his work of fixing advertisement plates to walls and can never persuade himself that they are securely fastened. He fancies the nails are bad, or the mortar loose, and makes himself ill over it. I have pointed out to him that if a plate fell it would almost inevitably slide down the wall. This has not prevented him from painting a most elaborate mental picture of the decapitation of an unfortunate youngster, who happened to be playing marbles with his head against the wall. To enumerate all his troubles would take a small volume. I have a great pile of his letters before me now, and I suppose they constitute one of the most extraordinary analytical autobiographies it would be possible to find. In reading them I cannot help marvelling at the strange unshapely wonder of such an imagination. He makes every incident in his life the foundation stone of a castle of fancies, and of late years each castle has become a prison—a torture chamber in which he has dissected his motives and his actions until he has ceased to believe in himself at all."

When I first saw this patient the "folie du doute" and "délire du toucher" were constant and most varied in their manifestations. If he accidentally touched persons in the street, he began to fear that he might have injured them and exaggerated the touch into a more or less violent push. If the person touched were a woman, he feared that she might have been pregnant and that he might have injured the child. If he saw a piece of orange peel on the avement he kicked it into the road, but soon afterwards began think this was a more dangerous place, as anyone slipping on might strike his head against the curb-stone, and so he was esistibly impelled to return and put it in its former position. one time he used to bind himself to perform certain acts, by ving he would give God his money if he did not do them. en sometimes he was uncertain if he had vowed or not. Owing this he gave sums to religious objects which were quite disportionate to his income. Apart from his peculiar fancies I d the patient perfectly rational and intelligent, and though "délire du toucher" hindered him greatly in his work, he generally managed to execute it, though on some occasions he was compelled to abandon the attempt. At this time I tried to hypnotise him on twenty-four occasions, but apparently without success and he was then compelled to leave town. He returned on April 2, 1895, for a week's further treatment and told me that since his former visit his morbid ideas had not been so frequent and marked and were accompanied with less mental agony His condition, however, leaves much to be desired.

(8) Mr. H., aged 33, tall, strong and athletic, first consulted me March 7, 1894. He stated that he had always been of a sensitive disposition and inclined to be morbidly self-conscious. Of late years this had greatly developed and made his life a burden to him. He had the fixed idea that he was constantly making mistakes in business and that all those with whom he was brought in contact considered him a fool. During a business interview he was embarrassed and unable to speak and felt that everyone must notice this. He had the same feelings in reference to society and shunned it as much as possible. He also had morbid and entirely unfounded fears about his physical condition Hypnotised ten times to July 11, 1894, when his morbid ideas had entirely disappeared. He has just reported himself, April, 1895, as well. If he has any anxieties about his business, he says, these are only natural ones and he has lost all his morbid fears and embarrassments. He is now fond of society and at his ease in it.

(9) Mr. I., aged 35, was sent to me by Dr. de Watteville on October 29, 1894. Illness commenced six months ago after the sudden death of his brother-in-law. He began to sleep badly and to dream of his own death. The insomnia continued and he was haunted by constant fears in reference to himself and his family. He dreaded that he would lose his employment and that he woul find his wife and children dead when he returned from work, & He became afraid of open spaces and was unable to cross t road without assistance. One day, when sitting alone in his p lour, he believed he saw two men bring his coffin in. He ne had a moment's freedom from his fears and was so miserable t he had strong suicidal impulsions. About this time he c menced to have frequent attacks of giddiness and felt that would fall down if he did not catch hold of something. On occasion only did he lose consciousness. He was hypnotised times up to November 12, upon which date his morbid fears almost entirely disappeared, but as he still had attacks of g ness and sometimes felt depressed, I continued to hypnotis

once or twice a week up to the present date (April 26, 1895). Present condition: He sleeps well, has had no giddy fit for two months, no agoraphobia or return of his other morbid fears, and deep hypnosis can now be easily and quickly induced.

(10) Mr. Watts. I was asked by Dr. Hack Tuke to try to hypnotise this patient, No. 10 in his list of cases. At this time he had been twelve months in Bethlem Hospital and was on the eve of being discharged, as his mental condition remained unchanged, and it was considered unadvisable to retain him longer. Dr. Percy Smith kindly consented to keep him another month for the purpose of this experiment, and I attempted to hypnotise him twenty-three times without success. I met him accidentally the other day; he told me his condition was unchanged and that he was going to live on a farm.

Dr. Hack Tuke also asked me to try to hypnotise No. 11 of his cases. When I saw this patient he was obviously insane and extremely suspicious, and nothing would induce him to submit to the treatment.

(11) [Liébeault.] Mrs. ——, May, 1892, aged 36. Has had good health until the present illness commenced eleven months ago. Her father committed suicide by drowning; her first husband was accidentally drowned twelve days after marriage, and she lost her second suddenly after she had been married three years. Since then she has avoided her friends, slept badly, neglected her work and her person, and has had the fixed idea that she would commit suicide by drowning as her father did. Hypnotised fifty-two times from May to July, 1892, when all ideas of suicide disappeared. In a recent letter Dr. Liébeault tells me that she remains well mentally and physically.

(12) [Schrenck-Notzing.] Mr. ——. Fixed idea regarding catching cold kept this patient for six months in his room. Hypnotised. Recovered.

(13) [Schrenck-Notzing.] Mr. ——, aged 24. Had periods of ror and uncontrollable sensations and ideas with the fixed idea at his *fiancée* did not love him. Hypnotised. Recovered.

(14) [Hecker.] Mrs. ——, suffered from diseased desire to wash self continuously and unnecessarily; had morbid fears that doors and windows were not properly fastened, and fancied she dressed herself untidily. On one occasion, after having t two hours vainly trying to dress, she sent for Hecker, who d her in a state of great anxiety, putting on and off her clothes exhausted. Hypnotised for a month. Recovered.

5) [Wetterstrand.] Mrs. ——, aged 42. For several years

had a morbid dread of thunder-storms; did not dare go into the country in summer, and in winter dreaded what the summer might bring. Constantly watched the appearance of the sky. Hypnotised twenty times. Recovered.

(16) [De Jong.] Mr. ——. Agoraphobia of many years' standing. Hypnotised ten times. Recovered. No relapse after a year and a half.

(17) [De Jong.] Mrs. ——. Fear of storms and of travelling by rail. Hypnotised. Recovered.

(18) [Van Eeden and Van Renterghem.] Mr. ——, aged 56. Psychical impotence for three years with the fixed idea, which he recognised to be false, that his wife wished to be unfaithful to him. Hypnotised eight times. Recovered. No relapse after twenty-two months.

This list might be largely extended. Many of the cases referred to at the commencement of the article were successfully treated by hypnotic suggestion, and many others have been reported by Bernheim, Delbœuf, Gorodichze, Russell Sturgis, Voisin, Burot, Mavroukakis, Bourdon, &c.

My chief difficulty in such cases has been the induction of the primary hypnosis. Various continental statistics apparently show that the majority of mankind can be hypnotised; Schrenck-Notzing's "First International Statistics of Hypnotic Susceptibility," published in 1893, give 8,705 cases by fifteen observers in different countries, with six per cent. of failures. Wetterstrand reports 105 uninfluenced out of 6,500 cases, and I formerly found patients drawn from my own practice equally susceptible.

For the induction of hypnosis the patient's attention is necessary, and in cases of imperative ideas, this is usually otherwise occupied as the result of the nature of the mental affection. Many also belong to the class the most difficult to influence, *i.e.*, the nervous and emotional. That such patien are the easiest to hypnotise is the conviction of all those w have no practical acquaintance with the subject, but this opini unfortunately, has no foundation in fact. Moll says: "T mentally unsound, particularly idiots, are much more difficult hypnotise than the healthy. Intelligent people and those w strong wills are more easily hypnotisable than the stupid weak-willed. Sex has no particular influence." Accordin Forel, "every mentally healthy man is naturally hypnotisal Mr. Wingfield hypnotised 152 Cambridge undergraduates at first attempt, while Esdaile found no difficulty in ind hypnotic anæsthesia among the coolies and felons of Be and these, as he justly remarked, were by no means nervous persons. The fact that many of my patients had read sensational newspaper and magazine articles on the subject did not render my task easier.

Personally I have never seen the slightest untoward symptom result from the skilled use of hypnosis, nor any evidence that its dangers, under these circumstances, have any existence save in the brain of the novelist. Forel says: "Liébeault, Bernheim, Wetterstrand, van Eeden, de Jong, I myself, and the other followers of the Nancy School, declare categorically that we, supported by the material of many thousands of hypnotised persons, have never observed a single case of mental or bodily harm caused by hypnosis, but on the contrary have seen many cases of illness relieved or cured by it."

As regards the connection between "folie du doute" and "délire du toucher" it is interesting to notice that the latter condition occurred in No. 7 as soon as the patient's doubts took a material form, but was not associated with it when his obsessions were purely intellectual. Does not this show that the appearance of the "délire du toucher" simply depends upon the nature of the "folie du doute," and that the incessant washings, &c., are the patient's natural physical efforts to rid himself of his material fears, while the character of the purely intellectual obsessions renders such relief impossible. G—— sought and found its nearest equivalent in telling his religious troubles to an older friend, in whose opinions he tried to find consolation. From this point of view the second of the cases quoted by Ladame shows "folie du doute" unassociated with "délire du toucher." This separation is, I think, more artificial than real. In his first case, that of the young man who frequently washed his hands through fear of contamination, it is by no means clear that this was a case f "délire du toucher" without "folie du doute." Surely the normal and incessant washing must have been the result of ubts which he thus tried to remove. The same objections ght be raised to van Eeden's cases where "folie du doute" is to have resulted from "délire du toucher."

Dr. Hack Tuke regards imperative ideas, and the acts result- from them, as essentially automatic and considers this r fundamental characteristic. Are these acts automatic? An matic act is simply an habitual voluntary one performed tentively or unconsciously; while the so-called automatic of the sufferer from imperative ideas are carried out in sition to his volition and frequently associated with intense and painful consciousness. Possibly with justice they might be called reflex, seeing that they are "fatal, unchosen, response to stimulation."

The fact that an imperative idea remains a stranger to the patient's ego distinguishes it, according to most authorities, from an insane delusion. This rule has its exceptions. One of my patients commenced ten years ago to be "inhibited" by various superstitions. At first this rarely occurred, but latterly, owing to the number of his unlucky days, &c., the performance of many acts is often interfered with. Many people, by no means insane, actually believe in, and are influenced by, identical superstitions. This patient, however, does not believe in them and keenly resents their interference with his actions. The non-assimilation of the imperative idea sometimes, then, constitutes the morbid element, and this apparently depends more upon the individual than upon the idea itself. The patient who made herself miserable about the Creation might, under other times and circumstances, have taken pleasure in discussing "the number of angels who could stand on the point of a needle, or whether, in passing from point to point, they had to traverse the intermediate space." The imperative idea to discover the site of ancient Troy only differed from those we are discussing in the fact that it was assimilated by its possessor, but this did not constitute insanity.

In nearly all my cases the condition appears to have had an emotional origin. The shock of the sudden death of a relative caused one patient to fear his wife would die, another dreaded travelling after being frightened by a drunken man in a railway carriage. Although in some instances the emotional element changed its character, and in all became greatly intensified, it was certainly often associated with the commencement of the original trouble.

Imperative ideas are usually regarded as being typical o
degeneracy and especially of hereditary degeneracy. Some o
my cases seem to confirm this, they were weak mentally an
physically and had unsatisfactory hereditary antecedents.
several instances their imperative ideas had become ins
delusions, many of them had suicidal impulses, one attemp
suicide and another had hallucinations. On the other h
the transition from the normal state to imperative ideas
almost insensible; the repetition of an insignificant saying be
according to Ribot, the slightest form, and pre-occupation,
as anxiety about an examination, a degree higher. Most chil
too have suffered at one time or another from imperative i

This, as a popular writer has justly remarked, appears to arise from an exaggerated sense of the importance of what they say and do, and also from an exaggerated fear regarding the notice taken of them by others. He says: "How miserable we sometimes make ourselves over some silly remark we have made. Some of us even keep a little store of foolish things we have said or done at various times—and take them out occasionally and blush over them. As a child I blushed for years at the thought of having piped out a response in church in the wrong place, before the clergyman's turn was over. I felt as if the whole congregation turned and gazed at me with scornful ridicule. As I walked away everyone who glanced at me I felt sure was thinking, "There goes the child who made that extraordinary squeak in church."

Everyone cannot have fixed ideas, as for example idiots, who possess little spontaneous and no voluntary attention, while, as Ribot says: "In every sound human being there is always a dominant idea which regulates his conduct; such as pleasure, money, ambition, or the soul's salvation." Some of my patients were physically far above the average, and many of them possessed mental endowments of high quality, and their morbid ideas did not prevent them doing valuable work. Most of them, it is true, were of an emotional, nervous type, but is the sensitive, mobile brain necessarily degenerate? May not the accidents to which it is liable be the result of its higher and more complex development? The thoroughbred is more emotional and nervous than the cart horse, but is this necessarily an evidence of its hereditary degeneracy? The term degenerate is applied so freely and widely by some modern authors that one cannot help concluding that they rank as such all who do not conform to some primitive, savage type possessing an imperfectly developed ervous system.

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J. MILNE BRAMWELL.

ON SYPHILIS OF THE NERVOUS SYSTEM.

[*With a Critical Digest.*]

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(*Continued from p.* 147w.)

APPENDIX TO PART III.

THE third part of this communication, published in the last number of BRAIN,[1] dealt with syphilitic disease of the spinal cord. It happened to be published a few days before Mr. Jonathan Hutchinson[2] brought forward the discussion of cases of nervous disease occurring early in syphilis, with especial attention to cases of syphilitic paraplegia during the secondary stage, or during what was assumed to be so. Incidentally, the *early* occurrence of syphilitic spinal cord disease had been the subject of considerable notice in my communication; as, *e.g.*, where syphilitic disease of large spinal veins and arteries, with the accompanying meningitis—which so often is of gummatous form—were taken "to explain such occurrences as local softening, hæmorrhage, sudden increase of pre-existent paralysis or paresis, or the onset of sudden paraplegia—perhaps at an early stage of the disease—perhaps even appearing almost like a precursory phenomenon" (p. 144) Or where it is stated that many cases of syphilitic paraplegia sudden or rapid in onset, attributed to myelitis, are probably due in reality, to the softening of portions of spinal cord followin upon occlusion of syphilitic blood vessels. Or where sudde flaccid paraplegia was found to be due to hæmorrhage into t spinal grey from syphilitic blood vessels with lepto-meningit Or where Gilbert and Lion's investigations of the pathologi changes in early syphilis of the spinal cord are stated in su mary. Or where the paraplegia is described as being, "less often severe acute palsy, perhaps from arterial thrombosis or from acute central myelitis, which usually is promptly fatal," and the softening or death of portions of the cord is described as being sometimes rapid or sudden.

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[1] BRAIN, Spring Number, 1895, p. 141.

[2] Roy. Med. Chir. Soc., London, February 26, 1895.

Mr. Hutchinson brought fifteen cases of acute paraplegia occurring between half-a-year and two years after the primary syphilis. But other similar cases, which he observed occurred later after infection. In general type, apparently, they only differed somewhat from Erb's; and did so in the rapidity or suddenness of onset, or of development, of the paraplegia, and the frequently severe degree of anæsthesia occurring at least temporarily. Like Erb in *his* cases he assumes them to be due to transverse dorsal myelitis, but had had no deaths. The cases with necropsies, cited from Gull, Fagge, Arthur Fox, Cropley and others, showed chiefly an acute myelitis.

In the discussion, Dr. George Ogilvie set forth a statistical array of examples of *early* nervous affections in syphilis, drawn from the writings of continental authorities.[1] Of these the most striking and most relevant to the point under discussion, is the experience of Goldflam, who "amongst eighteen cases of syphilis of the spinal cord found thirteen during the first two years." And Dr. Ogilvie rightly added that "cases of exactly the same nature, indistinguishable from the early ones in any way, occur late in syphilis."

Differing from the general type made by Erb, as well as from the more often acute (compared with Erb's) but non-fatal cases by Hutchinson, there is a severe and often fatal form of paraplegia. Usually an early phenomenon, probably it is not always ecessarily or essentially so, as it has been stated to be by Lamy.[2] Most often, according to him, it occurs within, or shortly follow-g the first year after the appearance of hard chancre. Most en sudden in onset, the paraplegia is generally heralded by a ort premonitory stage; such as pain in the spine, weariness, or esis, or bladder symptoms. The paralysis may come on in a ht, in a few hours, or suddenly. When the disease has formed, intensity of the symptoms is unusually great; absolute lysis of motion, and even of sensation, may occur in the

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Braus, 44 per cent. of brain syphilis within first year after infection. iac, 43 per cent. of cerebro-spinal syphilis within two years. Rumpf, cent. of brain and cord meninges syphilis within one year. Goldflam, cent. of spinal syphilis within two years.

De la Méningo-Myélite Syphilitique, p. 101."

parts affected, although not in all cases, for the anæsthesia may be incomplete or may be unilateral. The tendon-reflexes are diminished or abolished and then the limbs are flaccid. Electromuscular excitability may be lessened or lost, paralysis of bladder and rectum may be complete. Bed-sores tend to form. The usual lesion is an acute central myelitis. Or there may be luetic spinal lepto-meningitis, chiefly affecting the veins, and partly implicating the cord itself, in which latter, also, are secondary changes due to interference with the circulation, and consisting of colloid exudation in the grey, and areas of nerve-fibre degeneration in the white (Hayem, Julliard, Siemerling, Déjerine, Erb, Lancereaux and others).

PART IV.

SYPHILIS AND TABES: THEIR RELATIONSHIPS.

A.

The doctrine of the syphilitic origin of tabes dorsalis is mainly due to Prof Fournier,[1] of Paris, and to Prof. Erb,[2] of Heidelberg. But thirty years ago, in his work on tumours, Virchow asserted that, indubitably, many cases of tabes dorsalis—of grey atrophy—are syphilitic. Also, previously to Fournier and Erb, had Wunderlich mentioned, besides permanent paraplegias, tabetiform symptoms as due to spinal syphilis; and Berger[3] appears to have observed much the same thing.

Lancereaux[4] admitted the possibility of at least somethin analogous to tabes as a result of syphilis.

Erb,[5] in 1883, and in part previously (*loc. cit.*), declared syphi to be one of the most important, or *the* most important of conditions for the origination of tabes, and that a person not p viously affected with syphilis has scarcely any chance of bec ing tabetic. And, in 1892,[6] he gave 89 per cent. of tabetic having had previous syphilitic infection, as compared with twe two and a-half per cent. of other hospital patients who were not tabetic. Leyden,[1] in 1883 and 1892, took the opposite view.

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[1] "De l'ataxie locomotrice d'origine syphilitique," Paris, 1876.

[2] *Deutsches Archiv für Klin. Med.*, July, 1879.

[3] Cited by Prof. A. Eulenberg.

[4] "Treatise on Syphilis," *New Sydenham Soc. Trans.*, 1869, vol ii

[5] *Berliner Klinische Wochenschrift*, 1883, No. 32.

[6] *Berliner Klinische Wochenschrift*, 1892, Nos. 29 and 30.

But while Erb did not consider it proved that tabes is a specific syphilitic disease, or late manifestation of syphilis, Moebius[2] (1884) held empoisonment of the organism as necessary to tabes, admitting as the only efficacious poison—syphilis; and, doubtfully, ergot.

So long ago as 1876, writing on syphilis and insanity, in the part of the article devoted to gross syphilitic nervous disease, I[3] published, amongst other cases, one, the spinal element in which was spoken of as simulating tabes. (There were typical ataxy, Romberg's symptom, &c., and intercurrent strabismus and diplopia, also dimness of vision.) And it was so spoken of because, at that date, syphilis was not, or was scarcely recognised as in any direct ætiological relationship with tabes, *i.e.*, as productive of "systematic" spinal cord disease, and the mental recovery (at least for the time being) and the vast improvement of the tabetic symptoms, under anti-syphilitic treatment, led me, at the time, to conclude that the symptoms were scarcely due to true tabes but to ordinary syphilitic inflammation or neoplastic diffuse growth. *Now*, some would say it was a case of tabes, and probably it was the first case published in this country as being syphilitic.

The question of recovery, in this and similar cases, will recur after mention of two other cases of well-marked, typical, tabetic symptoms, in persons believed to have had previous syphilitic infection, on whom I made necropsies some years ago. Both were general paralytics. In one there was chronic spinal meningitis and thickening, particularly over the posterior surface of the cord. The posterior columns presented the typical grey degeneration and sclerosis of tabes, chiefly in the lower dorsal and lumbar egments of the cord, in parts of which the whole of the posterior edian and posterior external columns was affected, but, above, e lesion gradually becoming limited to the posterior median lumns, and invading the bulb. Many of the lower posterior nal nerve roots were similarly lesed. Some optic nerve rophy.

In the other, also, was chronic spinal meningitis, all the changes ut to be mentioned being confined, or nearly so, to the terior surface of the cord; namely, thickening of spinal dura

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"Real-Encyclopädie der Gesammten Heilkunde," 1883. *Berliner ische Wochenschrift*, 1892, Nos. 17 and 18.

ited by Eulenberg.

British and Foreign Med. Chir. Review, July 1876, p. 194-5.

and arachnoid; numerous white cartilaginoid plates in the layer of arachnoid next the cord; numerous adhesions of the meninges between themselves and to the cord, by distinct pseudo-membranous bands and filaments. The arachnoid thickened, opaque, semi-gelatinous in appearance. The posterior columns of the cord were lessened in consistence, altered in appearance, and the seat of myelitis taken to be of subacute type (numerous round cells, infiltrating profusely. Some scattered granule-masses).

Interesting in relation to the mobile recurrent right external strabismus (and to [?] the permanent slight right exophthalmos) was the subacute inflammation found affecting part of the length of the right motor oculi nerve near its root.

B.

Reverting to the recovery of apparent cases of tabes as bearing upon the question of their syphilitic origin, it has been held (as, *e.g.*, by Gowers,[1]), that as the symptoms of syphilitic disease of the nervous system depend, not on the specific process, but on the alterations it produces in nerve tissue, and as treatment exerts a direct effect on the specific process only, it follows that the persistence of symptoms, in spite of treatment, indicates persistence only of the damage done to the nerve elements, and not a continuance of the lesion producing the damage.

But dissipation of the specific process must in many, especially in early, cases permit the damage of nerve elements, still incomplete, to be repaired, or partly so; and it has been reported by Dinkler[2] that of Erb's cases of tabes 58 per cent. showed some improvement under anti-syphilitic treatment. And I have foun tabetic symptoms somewhat improved, and the associated menta disorder (for which they came under care), alleviated unde iodides. Sachs, also,[3] has found vigorous treatment with H and iodides benefit the ocular, ataxic, tendon reflex, and painf symptoms of tabes. And going back to my early student da although we saw tabetic patients treated with nux vomica, c nabis indica, anodynes, arg. nitr., and pot. iod., with or with actual cautery or blisters over the spine, yet a renowned clin teacher, Da Costa, taught us that the only drugs at that t apparently of any ameliorative effect on the disease—as dis guished from mere relief of pain, &c.—were iodide of potassium and nitrate of silver, so that the iodide is a very old friend in the treatment of tabes.

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[1] "Syphilis of the Nervous System," 1892.

[2] *Berliner Klinische Wochenschrift*, 1893.

[3] *New York Medical Journal*, Jan., 1894.

Some cases have been reported of improvement, of great improvement, or even cures of tabes[1] by anti-syphilitic treatment. For obvious reasons this is more likely to occur in private than in hospital practice. The patient is the more entitled to the chance of obtaining the possible benefits of specific treatment (Gerhardt) the nearer the onset of tabes is to the date of primary infection, the stronger and better nourished he is, and the more manifest are signs of syphilis. Moreover, the expectations of improvement under specific treatment are more favourable in the so-called anomalous or atypical examples of tabes, *e.g.*, cases with symptoms predominating on one side, presenting a less tardy course than the typical cases, and advancing by strokes. And Sachs suggests as a point sometimes helping to indicate cerebro-spinal syphilis simulating tabes (and, therefore, more curable than tabes proper)—an unusual irregularity of development of the symptoms; *e.g.*, the light reflex being lost in one eye very much earlier than in the other, or while the one is lost the other remains normal for years, or one or both pupils respond neither to light nor in accommodation, or any active syphilitic process at the brain-base is apt to be associated with or to long precede genuine tabes.

Are the alleged cures of tabes really examples of recovery from so-called pseudo-tabes? Syphilitic disease of spinal meninges with tabetiform symptoms may be accepted as one form of pseudo-tabes. And in "atypical" cases of tabes, instead of merely the ordinary typical tabetic sclerosis or grey degeneration of posterior columns and nerve roots, there may be a fresher and pecific syphilitic morbid process.

Dr. Adamkiewicz has had several cases, a very careful study f which has led him to the belief that there is a special form of bes having certain characteristic symptoms, and a specially fined clinical course, which is positively curable by *early* ti-syphilitic treatment.

"The symptoms of this curable form of locomotor ataxia : Ataxic paresis with loss of the tendon reflexes, paresis in the ribution of certain cerebral nerves, tabetic paræsthesia with- objective disturbances of sensibility, and pain especially on sure in the lumbar region. The clinical course of the disease is characterised by a rapid advance of the paresis of the lower extremities into complete paralysis, by ascent of this paralysis up to the lower part of the cervical spine, and, when the anti-syphilitic treatment is early begun and energetically carried out, by a final complete restoration to health. The duration of the disease, when so treated, is three or four months. The most important clinical symptoms of the disease—ataxia and motor paresis—point to an involvement of the pyramidal tract as well as of the posterior columns, and the fact of a cure by means of iodides and mercury, to functional disturbance resulting from a not irreparable process. There can be question here of syphilitic products only, which appear under the form of obliterating endarteritis and syphilitic pachy-meningitis.

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[1] Cure in one case lasting 18 years (Reumont).

"When, through lack of proper specific treatment, there is no resolution of the syphilitic products in the spinal cord, the diseased vessels become obliterated, and the parts of the cord supplied by them become sclerosed. In this case, similarly to what he has shown to occur in tuberculous pachymeningitis, wedge-shaped lesions are formed in the substance of the cord in which the nerves are destroyed. He has called this lesion, which results in incurable spinal paralysis, infarct of the cord."

The general belief that syphilitic vascular lesions of the brain only, and not of the spinal cord, are curable, is disproved, he said, by his observation.[1]

It is probable that most of us have erred in giving insufficient doses of iodides in syphilitic nervous diseases when heroic doses are needed. In cases of general paralysis and of tabes, apparently springing from syphilis, large quantities of K.I., or of Na.I., are given by some.

For example, Max Weiss[2] describes a case of advanced tabes, in which the connection between that disease and syphilis was very clear. "This case is especially noteworthy from the fac that a regular and steady specific treatment markedly an rapidly diminished pronounced objective and subjective tabet symptoms, a few of these even disappearing entirely. The trea ment consisted solely of rather large daily doses of iodide sodium, increasing from 5 to 8 grammes (1¼ to 2 drachms) several months. The patient was an engineer, 35 years o who had never suffered from illness during childhood; in 1883 acquired an indurated specific ulcer, with secondary symptoms. He was given twenty injections of corrosive sublimate and small doses of iodide of potassium; in July, 1884, a lingual ulcer developed, which underwent complete resolution after twenty-four injections of corrosive sublimate. Since that time there had been no specific eruption either on the body or the visible mucous membrane. In 1886 several attacks of nausea and vomiting occurred, each lasting about fourteen days, accompanied by severe pain in the back. In the autumn of 1887 renewed attacks of vomiting occurred early in the morning (gastric crises). From 1889 there were almost daily attacks of vomiting. Nutrition was much impaired and the body-weight decreased. He suffered from lancinating pains over the entire surface of the skin, more particularly in the arms and legs, most frequent after a change of weather." In 1890, static and dynamic inco-ordination was first noticed, with paræsthesia of the toes, soles of the feet, and the two small fingers of each hand, diminished tactile sensibility in the epigastric region, and fatigue, after the least attempt at walking. "Constipation, cramp-like pains in the abdomen (intestinal crises), some retention of urine, and severe boring pains in the urethral canal were added to the other symptoms. In the spring of 1893 the sight was poor at a distance of from twenty to thirty steps, but there was no trouble in reading and writing. In August, 1893, the patient was submitted to a systematic iodide treatment. For the first two weeks he took daily 5 grammes (1¼ drachms) of diluted iodide of sodium, no symptoms of iodism being observed. The daily dose was increased 2 grammes (31 grains), and for some time 3 grammes (46 grains). Within a month the daily gastric crises ceased suddenly, and have never since reappeared; in September isturbances of co-ordination diminished perceptibly, and in ctober, when the patient was taking 8 grammes (2 drachms) of dide of sodium daily, and had already consumed 500 grammes pound) in all, without any untoward symptoms, no evidences ataxia were present.[1] The cloudiness of vision had also dis- eared, the lancinating pains occurred but seldom, and were h less severe than formerly. The patient, even after walking ral hours, did not feel any fatigue. His appetite has greatly ased, and his weight has increased 6 kilos (12 pounds). tment is still being continued in daily doses of from 6 to 8

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[1] Int. Med. Cong., Rome, *Medical Record*, May 5, 1894.

[2] *Centralblatt fur die gesammte Therapie*, February, 1894. Abstract *Univ. Med. Journ.*

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urity of the iodide of sodium employed is an essential condition of such ent.

grammes (1½ to 2 drachms), with short intermissions. The paræsthetic symptoms have almost entirely disappeared." The urethral crises and the weakness of the detrusors persisted longest, and galvanization through the lumbar cord, perineum and bladder, was resorted to, with internal administration of ergot and strychnine, the result being that for three weeks before the time of report the patient no longer complained of urinary disturbance or of pain in the urethra.

Weiss refers to a similar case treated by Werner Stark. The patient was a woman, aged 43, who had become infected thirteen years previously and recovered without relapse. Eight years later the first indications of tabes appeared, the symptoms increasing in intensity until the patient was unable to walk. "No disturbances of the digestive or urinary organs occurred. When the patient was first seen by Stark, in 1890, she was pale and thin; there was complete ataxia of the legs, analgesia and partial anæsthesia of the skin, as well as weakness and atrophy of the muscles of the legs; the patellar reflex was absent. Psychic and ocular disturbances were not present. The patient had been discharged from hospital as incurable. Stark first gave 50 grammes (1⅔ ounces) in 400 grammes (13 ounces) of water, a tablespoonful being taken three times daily. After some time the pains became less severe." The solution was then strengthened to 60 in 400 grammes (1⅞ to 13 ounces); after three months to 75 in 400 grammes (2½ to 13 ounces); and again after three months to 100 in 400 grammes (3¼ to 13 ounces), four tablespoonfuls daily, this strong solution being taken for four months. "After the first increase of the dose improvement soon occurred, so that the patient was able to do a little light work; after the second increase the pains disappeared and the ataxia and anæsthesia decreased. After the last increase, when the patient wa taking 12 grammes (3¼ drachms)[1] of the iodide of potassium daily there was perceptible improvement; the anæsthesia and atax disappeared, and the muscular atrophy diminished; the patie could walk quite well with the aid of a cane or support. Dur the entire time she did not suffer from any disturbances of [illegible] digestive or other organs. Sometimes the treatment was [illegible] tinued steadily for weeks, and again it was interrupte intervals; during these, however, Stark observed that impr ment was not progressive, and that there was occasiona tendency to relapse. At the time of writing, the patient [illegible] for a year only, occasionally been taking the iodide of potas [illegible] feeling stronger after each treatment."

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[1] Fifteen grammes (4 drachms) ?

C.

From the third Part of this communication, namely, that on syphilitic spinal cord disease generally, we carry forward a reminder of the cases linking tabes with spinal cord disease clearly due to the specific lesions of syphilis. Such, especially, are the cases of syphilitic lepto-meningitis invading the cord, particularly its posterior columns and horns, sometimes producing a more or less tabetic, or quasi-tabetic, posterior sclerosis, and diagnosed during life as examples of tabes, or of anomalous, atypical, or pseudo, tabes. Of such kind are cases therein cited from Ewald, Eisenlohr, Kuh, Dinkler, Brasch, Schulz, Siemerling, and one or two of the present writer's cases.

Moreover, syphilitic spinal lesions have been found associated with the recognised lesions of typical or classical tabes. In this communication (see Parts I. and III.) have already been described in abstract two such cases, one from Raymond, and one from Hoffmann, and the second of the present writer's three cases, mentioned in this Part IV., was probably of that nature.

Here another may be added from Sachs.[1] "Clinically it was a typical example of tabes dorsalis."

Posteriorly, the spinal pia was covered by a thick gelatinous mass, from lower cervical to upper lumbar segments. The pia was thickened both in front and behind, and of leathery consistence. Microscopically, was diffuse sclerosis of posterior columns, extending from the lower cervical region to the lumbar enlargement, but best marked in the cervical and dorsal segments, and it included Lissauer's tract. In the lumbar segments, only the posterior portion of the posterior columns was affected much, the rest but little. "A few cells of Clarke's column could be made out only in the lower dorsal segments. The sclerotic issue differed in no respect from the classical order of spinal clerosis." Nerve fibres of cord elsewhere, and anterior grey atter, normal. In lumbar and dorsal segments was a wide- ead meningeal infiltration; also, there, the pia was firmly glutinated to the cord, particularly in the posterior and lateral umn regions; there, also, the posterior nerve roots were enerate, and the small blood vessels showed changes cha- eristic of syphilis. These changes were most in the lumbar on, where the sclerosis, on the contrary, was least. The pia "characterised by an invasion and proliferation of round , and by very typical changes in its blood vessels, large and l."

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[1] *New York Medical Journal*, January 4, 1894.

Consequently, besides posterior spinal sclerosis was a syphilitic lepto-meningitis "invading the cord at different levels, and occasionally invading a distinctly sclerotic area, also a very typical specific arteritis. Whether it was an endarteritis or a mesial arteritis cannot be determined easily," but in the majority of the vessels the innermost coat had undergone the greater changes.

The condition was taken to be a syphilitic process, superimposed upon the sclerosis, the sclerosis coming first, the syphilitic infiltration later on. So far as it invaded areas already occupied by sclerosis it would not occasion new symptoms. It is possible, or even probable, that the blood vessel change preceded the sclerosis.

A case of typical tabes dorsalis with central gliosis, in a syphilitic subject is reported by Nonne.[1] Besides the lesions of both conditions, were a recent focus of softening, explaining the paralysis of upper limbs; old ulnar neuritis; chronic cerebral lepto-meningitis; syphilitic endarteritis and mesarteritis of basilar.

In reference to cases of these kinds, besides what has already been stated, we shall notice presently that not only may myelitis or gumma be followed by tabes, but also that myelitis may come on in the course of tabes.

Clinically, also, we, in fact, get at least a close approximation to, perhaps an identity with, several clearly defined clinical entities according to the part or parts of the cord predominantly affected by syphilitic lesions. If these parts are the posterior columns (nerve roots and horns), the clinical image of tabes presents itself. If the postero-lateral columns (including crossed pyramidal tracts) are chiefly affected, and the posterior horn also somewhat invaded, the posterior columns less affected, th posterior root zones and nerve roots suffering little; then v get the symptom-group of Erb's so-called "syphilitic spi paralysis." If the posterior, pyramidal-tract, part of the late columns chiefly suffered, there would be a symptomatic app ance as of primary spastic spinal paralysis, but in such c other degenerations would perhaps be present, as may b ordinary primary spastic paralysis, namely in Goll's and dach's columns, and direct cerebellar tracts.[2] If, as occurs ra the anterior horns suffer most, there are, with other symp the phenomena of spinal progressive muscular atrophy, or, more acutely, anterior poliomyelitis; perhaps or possibly still more acutely, that form of it with, clinically, the acute ascending spinal paralysis (of Landry); if, with the degeneration of nerve cells of anterior horns, there is also predominant change in the lateral pyramidal tracts, the clinical aspect will be that of amyotrophic lateral sclerosis. Syphilitic cervical spinal cord change may simulate syringomyelia or Morvan's disease; and the syphilitic incidence chiefly on many of the posterior spinal nerve roots occasions a special symptom-group, as in cases by Kahler and Buttersack.

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[1] *Archiv für Psychiat.*, Band, xxiv. H. 2.

[2] Ad. Schule, *loc. cit.*

Likewise, the image of ataxic paraplegia may be the manifestation of syphilitic disease affecting both the posterior and the lateral columns, although such cases, finally, are apt to pass into a more spastic phase, and present the symptom-group of the spastic condition, already fully discussed in the Third Part of this communication.

But usually the lesions are multiple, partly diffuse, and partly concentrated in place, and the symptomatology correspondent thereto is mixed, combined, multiple; the disease being more or less widespread, yet selective of loci—selective, yet having a variety of affinities or tastes, so to speak.

In some of these cases of doubt whether there is genuine tabes; or, on the other hand, spinal syphilis, that is to say, in the sense of syphilitic morbid processes; the symptoms point to tabes, but there occurs a return of the knee-jerks which had been lost, or a restoration of equality between them on the two sides which had failed; and in some instances a cessation of external ocular paralyses, which had previously accompanied the failure n patella-tendon reflexes.

The fact is also to be borne in mind that certain cases of bes (contrary to what has been held by some) present not only typical lesions, but also degeneration of the comma-shaped dle more distinctly than of the surrounding parts. Or cases present degeneration of the antero-lateral ascending tract; f the direct cerebellar. In fact, the complications attending, etimes, the lesions of genuine tabes are numerous.

D.

e may now turn to the studies of some observers on the rtion, per cent., of tabetic cases presenting a previous y, or indications of syphilis.

Dr. Gowers,[1] in private consulting practice, found 58 per cent. of male cases give a history of hard chancre or of secondary syphilis. Two-thirds, he thinks, would be nearer the truth, could all facts be ascertained. In women he found antecedent syphilis to be traced rather less frequently than in men. "In the cases of tabes that succeed syphilis, the lesion is not syphilitic in histological character. In these, as in the cases in which syphilis can be excluded, the disease is a degenerative one. It is, moreover, rarely influenced by anti-syphilitic treatment. Hence it must be regarded as a degenerative sequel of syphilis rather than as a true syphilitic disease. . . . In a few cases, perhaps 10 per cent. of the whole, syphilis can be excluded with confidence. The causes operative in these can be traced also in some of the patients who present a history of syphilis, and in such cases the causation of the malady is probably complex."

Such other causes are spinal injury, exposure to wet and cold, excessive fatigue and over-exertion, acute diseases, *e.g.*, rheumatism and enteric fever, alcoholic excesses.

In the second case of tabes ever seen by the writer, excessively hard work and over-exertion, six years previously to the time the patient was seen, were apparently the (or the chief) ætiological factors.

Fournier has stated that over 90 per cent. of persons suffering from tabes have incurred syphilis. Yet in only 76·7 per cent. of these (112 in 146) was antecedent syphilis alleged to be indubitable. On the other hand, Borgherini[2] has recently collected statistics in Italy, according to which scarcely 30 per cent. of those with tabes were syphilitic. In A. Fraenkel's experience, the percentage was 50·7 per cent.; in Senator's and Mendel's 70 to 75 per cent. G. Lewin denied these high percentage Virchow considers the relations of tabes and syphilis not decided, and notes the rarity of visceral syphilis in tabes.

Of 102 cases of tabes, Gerhardt[3] concluded that 51, or ex half, were persons previously syphilitic. The *average* int between the time when syphilis was incurred and the on tabes in these cases, was found to be ten years; from col statistics, another places it at eight and a-half years.

Compare with this the *average* interval of only four ye examples of spastic spinal paralysis, taken to be syphilitic.

Yet the same observer speaks of seeing syphilitic spin disease supervene at from three months to twenty years after primary infection, and take similar forms under such very dissimilar time-circumstances.

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[1] "Diseases of the Nervous System," vol. i., 1886, p. 288.

[2] *Medical Record*, May 5, 1894.

[3] *Loc. cit.*

On the other hand, in 17 cases of insular sclerosis, no mention of syphilis was found.

Prof. A. Eulenberg[1] discusses "syphilitic tabes" in the pathologico-anatomical sense, *i.e.*, the occasional appearance of syphilis of the spinal cord under the clinical form of tabes dorsalis. He published brief particulars of 125 cases of tabes diagnosed with certainty, all other examples, he says, being excluded. Of these, 19 were women: for reasons given, he excepts them from consideration, as to the ætiological factors; but it is to be noted that in only one of them was syphilis certain; a few others were sterile, and one had a husband probably tabetic, points which raise a suspicion of syphilis. But the majority of the women were certainly not syphilitic.

Of the 106 male cases, 39 had some venereal infection; but of these, 11 had had only simple or "soft" venereal sores or chancres, without subsequent appearance of general syphilis. These, I think, we must exclude. The remaining 28 had had typical hard chancre, and general constitutional syphilis. These we may accept as syphilitic, and these only. Thus, for male tabetics there would be 26½ per cent. (nearly), previously affected with syphilis. (The percentage would be 36⅓, if we include, as Eulenberg does, the eleven cases with simple, soft venereal sores, and no subsequent external, or constitutional or general manifestation of syphilis.)

Neuropathic heredity was observed in 12 per cent.

Atmospheric *noxæ* (chills, wettings, &c.), in more than ne-third.

Severe bodily exertions in one-fifth.

Severe depressing emotions, previously, in 13 per cent.

Debilitating conditions, leading to general weakness and ex-stion, in 10 to 11 per cent. (*e.g.*, pulmonary, intestinal, ovario-rine, or malarial diseases; sexual and alcoholic excesses).

Traumatic damages in about 5 per cent.

Deformity of vertebral column in a few cases.

Previous acute disease in 5 cases (typhoid fever 4, dysentery 1).

Very often two or more of the above alleged factors were sup-d to have acted conjointly.

aking persons affected with all forms of nervous disease, collectively, Minor[1] found a syphilitic history about five times as frequent in Russians as in Russian Jews. In the latter, syphilis is very rare, owing to alleged reasons, which are duly set forth. And he found tabes about five times per cent. more frequent in Russians than in Russian Jews, and general paralysis about seven times more frequent. And yet to nervous diseases generally, the Russians are *less* obnoxious than are Russian Jews

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[1] *Archiv. für pathol. Anat.*, Bd. xcix., Hft. 1, p. 18.

In eight tabetic women he found seven certainly syphilitic, and the remaining case probably so.

The combined statistics in Russia (his, Korsakoff's and Kojewnikoff's), if correct, show that, among those affected with various nervous diseases, there were :—

In 4,700 Russians, 137 cases of tabes, or 2·9 per cent. (29 per 1,000).

In 696 Russian Jews, 6 cases of tabes, or ·86 per cent. (8⅗ per 1,000).

These combined statistics thus show the racial proportion, per cent., to be only about (not 5, but) 3⅓ times greater in the former than in the latter race.

Of the Russian tabetics, syphilis was believed to have existed in 60 per cent., for certain; and probably in some more. Of the eight Russian Jewish male tabetic cases he had known of (partly in above statistics), seven are stated to have been syphilitic.

The time between primary infection with syphilis and the onset of tabes, was found by A. Eulenberg to vary from either one or two years to twenty-nine years. Isaac cites an interval of from one to twenty-five years.

The last named[2] gives importance to the old objection that, whereas tabes is a system-disease of the cord, syphilitic disease, on the contrary, is characterised by interstitial or tumour-formin processes, so different from the primary parenchymatous morb processes affecting the nerve elements in tabes, and holds th the statistics have too many sources of error to attain certai on the ætiological connection between syphilis and tabes; t there is not an ætiological relation between them; but syph like other influences leading to tabes, may act by enfeebling a posed nervous system; and that anti-luetic treatment is con indicated in tabes.

Too often, we think, the statement of a pre-existent syph or even a simple venereal sore, real or supposed, has been t as sufficient proof of the existence of syphilitic infection, and, impliedly, of the syphilitic origin of a subsequently existing tabes. The same objections hold here as do also regarding the similar statistics in relation to general paralysis (see Part I.).

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[1] *Archives de Neurologie*, t. xvii.

[2] *Berliner Klinische Wochenschrift*, April 16, 1894.

Some of the errors into which observers and compilers of statistics have fallen, in this matter, are referred to by Hr. G. Lewin.[1] One is that genital scars have been taken as indication or proof of antecedent syphilis; whereas a soft venereal sore usually leaves a cicatrix, but a true hard one (sclerosis) only does so very rarely.

Moreover, and this is another source of error, a soft sore may become indurated, and such non-syphilitic hard (hardened?) chancres he designates, in their several kinds, as (1) follicular, (2) cicatrised, (3) artificial, and (4) phlegmonous. It is unnecessary to point out how misleading, therefore, may be a history of a hard sore.

Another error concerns the diagnostic difficulties as to the rashes, the ophthalmias, &c., of syphilis.

Following Virchow, he has found atrophy or aphasia of glands of the tongue, a valuable indication of syphilis in several cases where other specific lesion was scarcely to be found.

Blaschko emphasizes the importance of examining the patient's children when the personal examination fails to reveal any clear indication of tertiary syphilis, such as gummatous orchitis or smooth atrophy of tongue-root. In the children, "Hutchinson's teeth," and similar dental anomalies, are important for diagnosis of the parental malady.

E.

Is the morbid process in tabes proper due to syphilitic infection, including here, not only the spinal, but also the cerebral elements of tabes?

The posterior horns and columns, including Lissauer's tract nd the root zones in postero-external columns, in which tabic hanges first appear are quasi-accessories to the spinal cord, and present, quantitatively and qualitatively, the entrance of posrior root fibres at the various levels of the cord. So that, in e sense, tabes appears to be a degeneration of posterior roots they enter the grey and white of the cord.

As to the question whether this degeneration is *primary* or not, Sachs reminds one that degeneration of nerves occurs if they are cut off from the influence of their trophic nerve-cells. For the sensory posterior nerve roots and fibres, these cells are in the spinal ganglia on posterior roots, and in the terminal nervous end organs, chiefly in skin and musculature. Sufficient disease in those ganglia or in these bodies, or lesion of the nerves in their course, may give rise to the degeneration; ascending degeneration of sensory fibres in the cord would therefore follow (*inter alia*) a lesion between the spinal ganglia and the posterior horns of the cord. This lesion may be set up by syphilitic spinal meningeal disease; explaining a relation between the morbid anatomy of syphilis and of tabes. Thus further questions arise: (*a*) whether the syphilitic poison acts through the medium of the blood vessels, (*b*) whether the syphilitic poison brings about changes in the blood, and, if so, (*c*) whether these hæmatic changes are the cause of the changes in the walls of the blood vessels.

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[1] *Berliner Klinische Wochenschrift*, 1894, p. 657.

Ergot, a potent agent on the vascular system, also occasions a so-called form of tabes.

F.

We must now briefly refer, more pointedly, to the intoxications of the organism—its toxic or, toxæmic states—whether self-produced, or coming from without—whether chemical, or the results of perverted metabolism, or of microbic origin. These states are apt to manifest themselves by organic changes in the nervous system, and preferably by alterations either in the peripheral nerves, or in the posterior spinal columns, or in both. Thus, posterior columnar spinal changes are found in many infectious microbic diseases; in some maladies consisting of alterations in the constitution and life of the blood, however there may be produced; or consisting in perversions of metabolism, and of excretion; or are found in systemic intoxications which are to be directly connected with prejudicial drinks and foods.

Thus, like the peripheral nerves, the posterior columns appe to be particularly vulnerable in toxæmic, or exhausted cachect states.

As to the relations between syphilis and tabes, and the questi of toxæmia, the former are brought out more clearly in cases tabes in the upper classes of society, in gentlewomen, and children with hereditary syphilis.

If we admit a connection between syphilis and tabes, of what kind is it? That, at present, we cannot accept tabes as simply or absolutely a tertiary syphilitic disease is the view of Professor Strümpell;[1] and that, to form a more exact conception of the kind of connection will assist one to meet objections raised against the very existence of a connection. The most important of these objections are two, namely: (1) first, the often alleged total or comparative failure of anti-luetic treatment to control or to cure tabes; and (2) second, that the pathological anatomy of tabes is not akin to genuine syphilitic changes, but fundamentally represents a simple degeneration of some nerve fibre tracts. It is unsatisfactory to meet these objections by over-estimating the effect of anti-luetic treatment in tabes, and by deducing the morbid anatomy of tabes from vascular disease. A more satisfactory basis on which to take stand, is the recognition that tabes after syphilis would form no isolated phenomenon in pathology, but would fall into the large group of nervous consecutive diseases or lesions following infections of the most different kinds. For, after diphtheria, typhus, dysentery, and many similar infections, degeneration may affect definite portions of the nervous system—degenerations which are secondary simple degenerative processes, and not specific primary anatomical changes. In paralysis after diphtheria and typhus we do not find croupous inflammation, or typhous new formation, in the degenerated nerves. So, in chronic infectious disease, as, for example, in tuberculosis, there may be peripheral nerve degeneration.

Just as ataxy may be a nervous disease, or sequel consecutive to diphtheria, &c., so may tabes be a nervous disease, or sequel consecutive to syphilis. Possibly a persistent chronic slight intoxication by a chemical poison, occurring in the once-infected organism, may induce gradual atrophy in definite lines of nerve fibres.

And, continues Strümpell, the same holds good of general paralysis as of tabes; nearly related, they are in a certain measure, only different localisations of the same morbid process.

As to the above subject, some accept the general principle that syphilis is very important in the production of tabes; others support these views more or less, and either indirectly or directly. For example, concerning the alleged comparative non-effect of nti-luetic treatment, as ordinarily used, in tabes, it has been eld by Fürstner that the failure to cure grey degeneration of the osterior columns by anti-luetic treatment, is paralleled by the fact that diffuse diseases of the central nervous system of specific character are usually not ameliorated by anti-luetic treatment.

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[1] Versammlung deutscher Naturforscher und Aerzte zu Heidelberg, Sept., 1889.

On the other hand, objections have been made. Thus, Prof. Schultze, of Bonn, demurs to Strümpell's analogy between tabes and diphtheritic paralysis, for the latter, like ergotin-tabes, is distinguished from genuine tabes, inasmuch as they are regressive, but tabes progressive. In syphilis, there must be a persistent formation of its toxine, against which anti-luetic medication is powerless. Lead-paralysis would be a better analogue of syphilitic tabes than post-diphtheritic paralysis is; for in spite of cessation of the ingestion of lead, the disease often advances.

And as regards the assertion that tabes and general paralysis are so closely akin, or that they are even in a measure only different localisations of the same morbid process, it has been urged by Mendel that, although syphilis is one of the most important predisposing causes of tabes, yet tabes and general paralysis are not to be taken as being related in the way just mentioned; very few general paralytics having grey degeneration of the posterior columns; about 18 or 20 per cent. having loss of knee-jerks; only a very small number, proportionally, of true tabetics becoming general paralytics.

Tuczek affirmed that posterior column changes in general paralysis, do not always (he might have said, do not often) affect the posterior root zones. Lateral column changes mostly coexist.

H. Schüle's experience is, that so-called classical general paralysis is not so often or so certainly in connection with syphilis as tabes is.

That the connection between syphilis and tabes is much more simple than that between syphilis and general paralysis is held by Fürstner. Posterior columnar disease, alone, is not frequent in general paralysis; more frequent is that of posterior and lateral, or of lateral columns only. In syphilitic general paralysis the complicating spinal cord disease does not always invade the posterior columns.

Recurring to the asserted existence of a close kinship of tabes to general paralysis, or that they are different localisations of the same morbid process, I think we must bear in mind that (as compared with tabes) syphilitic general paralysis is an earlier manifestation of syphilis; is of a more active type; has indeed, usually, irritative inflammatory, exudative and degenerative elements—new formation, destruction, and degeneration, often simultaneously occupying the tableau—is less chronic and slow than tabes; often, indeed, is subacute; is a primary condition (as a rule) even partly in its degeneration. Whereas, in tabes, the grey degeneration is somewhat analogous to, or partly identical with, secondary Wallerian degeneration (degeneration of nerves consecutive to various lesions, injuries, &c.). But probably it is more precisely akin to peripheral neural degeneration or neuritis, from the toxines of infectious diseases, or from chemical poisons—in a general word; from toxæmic states, including infections, dietetic and chemical poisons, ptomaines, leucomaines; but herein it shows close kinship with general paralysis.

Therefore I think that tabes may be accepted as at least mainly of toxæmic origin, whether from:

Auto-intoxications, altered metabolism; or from

Chronic infections; as syphilis, tuberculosis, leprosy; or from

Acute infections, possibly; (such as diphtheria, typhus, enteric fever, influenza, variola); or from

Ergot—lead—Hg. ?—I. ?—CO. ?

Lithæmia, gouty and rheumatic blood-states generally, diabetes, anæmia perniciosa. (See also auto-intoxications, above.)

In a preceding part of this article, I have mentioned the assertion of some that the lesion of true tabes is only deuteropathic, is only a secondary, a consecutive change in the neuropathic sense. Undoubtedly, in many examples it is a secondary consecutive lesion in the neuropathic sense, and, in such cases, often consecutive to syphilitic disease, sometimes to other ætiological factors. For instance, syphilitic meningitis or gummatous infiltration, or gumma, may give rise to an ascending secondary degeneration or sclerosis; and so of other conditions, such as implication of the spinal ganglia on the posterior nerve roots in some morbid process, however set up; or from peripheral sensory-nerve neuritis or degeneration invading the spinal ganglia, whence, possibly, the morbid process may restart to the cord by way of the posterior nerve roots.

But in many cases of tabes there are not—or not such marked—indications of preceding pachy- or lepto-meningitis, myelitis, gumma, or implication of spinal ganglia or peripheral nerves, or disease of nervous sensory apparatus in skin and muscles—as to explain the posterior nerve root and posterior column change, satisfactorily, as a secondary neuropathic lesion. And the only explanation that commends itself to me, of the tabic lesions occurring primarily, is that there is a selective action upon and of the nerve fibres primarily affected by and to poison (toxine, &c.) circulating in the blood chronically, much as occurs in nerves in other toxæmic conditions.

The degenerative tendency effective alone may be determined in its operation by local disease. As examples of this, one author cites that set up by a cold bath or by a severe fall, very soon followed by loss of power, undergoing improvement or recovery, but succeeded by the ataxy of tabes.

An ascending neuritic or degenerative change, arising from the seat of primary infection on the genitals, and passing to the spinal cord, whether set up by a toxine, generated locally at first, or coming as an irritative-degenerative process extending upwards by continuity of tissue, is an alternative possibility, to which there are obvious points of demur. In fact, also, the cord and nerve root alterations would *not be primary* in such case.

G.—*The Connection between Tabetic-Arthopathy and Syphilis.*

Two examples are described by Dr. N. Muchin.[1]

The first, a ship captain, aged 41, had incurred primary syphilis eight years previously to his coming under the reporter's notice, and two years after that infection he felt the first evidences of spinal disease. Anti-luetic treatment partially relieved these, but in less than a year the arthropathy of the left foot made appearance. This was greatly ameliorated, indeed practically cured, by specific treatment. Syphilitic spinal paralysis of Erb's type had become fully developed. Clinically the arthropathy was manifestly of nervous origin; the effect of specific treatment, and the clear traces of syphilis, show its syphilitic nature.

The second patient, a merchant, aged 53, had incurred syphilis eighteen years before coming under the reporter's observation. Treated by inunction (*Hg.*?) he felt no further effects for nearly fourteen years, but then the symptoms of tabes dorsalis began. Two and a-half years later, the arthropathy of the left foot began. This recovered under specific treatment.

Muchin concludes that:—

In subjects of tabes, syphilis of the joints may in no wise differ from Charcot's joint disease.

A causative connection between Charcot's arthropathy and disorders of innervation of the joints may be taken as proved.

Yet, not rarely, there are arthropathies which are not of pure nervous, but of mixed, origin; wherein the disorders of innervation only serve as predisposing factors. These impair the nutrition of the joints, and form a particularly favourable ground for the action of external and internal noxious agencies.

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[1] *Deutsche Zeitschr. für Nervenheilkunde*, Bd. v., S. 255.

The most important of such *external noxæ* are mechanical injuries and chills; of *internal*, the chief are syphilis and "arthritismus."

Synovitis of knee-joint may be a manifestation of hereditary syphilis in the young; giving rise to comparatively moderate or little pain or difficulty in walking, and remediable by anti-luetic treatment.

PART V.

"SYPHILITIC EPILEPSY."

A.

If convulsive seizures come on for the first time in a person above 30 or 40 years of age, and otherwise in good health, the chances, according to Fournier, are eight or nine out of ten that the seizures are of syphilitic origin. So-called "epilepsy" is frequent in cerebral syphilis, the explanation of which lies in the fact that the lesions of the latter so often affect the several meninges, or some of them, and the grey cortex.

In cerebral syphilis of "epileptic form," what are the clinical aspects of the "epileptic" seizures? Fournier, while admitting that the "epilepsy" of (tertiary) syphilis is different in nature as a morbid entity, from true, essential, spontaneous, common, "primitive" epilepsy existing from birth or early youth, holds that, *clinically*, the two are much alike, and more so than has been alleged by some; and that, clinically, "syphilitic epilepsy" is a true epilepsy, and takes the forms of ordinary epileptic *grand mal* and *petit mal*, the former being, of the two, much the more common. For convenience of discussion, will here (under section "A") be followed the lines of his description.

Invasion of "Syphilitic Epilepsy."—In a few cases the "epileptic" phenomena, sometimes found in cerebral syphilis, come on alone. Especially may this occur at the onset, and for a limited space of time. But in the great majority of the cases they are accompanied or preceded by other cerebral symptoms, also of specific origin.

As to the time at which they appear:—Sometimes they are the first manifestations of cerebral syphilis, the earliest symptoms to appear; sometimes they come on later, and only after other symptoms, due to the same lesions, have been established; and sometimes they only first appear shortly before, or at, the closing scenes of the malady.

Subsequent Evolution of "Syphilitic Epilepsy."

This is summed up in three chief points:—

One is, that at first the bouts of seizures, identical with or analogous to each other, nearly always occur at moderate or considerable intervals; but later on tend more and more to occur at shorter intervals. The attacks are preceded in a minority of instances by various mental or sensory prodromata, especially headache, which in some is fixed and circumscribed; and in a few are preceded immediately by auras.

The second point is, that the epileptic form of cerebral syphilis, at its onset and for a time thereafter, may be unmixed, unassociated with other specifically originated cerebral symptoms, and, very exceptionally indeed, this purely "epileptic" clinical aspect may last a very considerable time, and the case may therefore readily be mistaken for one of ordinary epilepsy.

But, ordinarily, and this is the third point, the convulsive symptoms become subsequently modified by, mingled, associated or complicated with, either the symptoms ranged under the name of *petit mal*, or with cerebral symptoms of some other kind, or with both. For the attacks of "syphilitic epilepsy," at first far more frequently of the convulsive *grand mal* type, may subsequently present seizures of one or more of the various kinds grouped under the *petit mal* type. Or, again, the "epileptic" bouts may become associated with cerebral disorders of some other kind, and thus cease to hold the chief or sole place clinically, becoming only one feature in a more complex clinical aspect. The cerebral disorders, one or more of which may thus come to complicate the "epilepsy," are chiefly of three groups; namely, the cerebral congestive, the mental, and the motor (paresis, paralysis).

In the above, Fournier's view seems to have been open to exception on two points: the one in making epilepsy, in the *adult*, from *acquired* syphilis in its *tertiary* stage, show, at first, in almost all cases, the clinical aspects of true essential epilepsy (general convulsion, loss of consciousness); whereas, it is believed, it more often is a "partial epilepsy" at first; the other (p. 186-7) in making the change from the clinical aspect of "essential" or true epilepsy, to that of "symptomatic" epilepsy (if, and when, it occurs) to be due only to the adjunction of cerebral symptoms of other orders, whereas the features of the attacks themselves assist in the differential diagnosis. Indeed he, impliedly, in part makes good this omission later on.

How, then, is "syphilitic epilepsy" to be diagnosed from ordinary true epilepsy? Does its present special peculiarities in (1) the mode of appearance and succession of its seizures; or (2) in the symptoms constituting those seizures; or (3) in the conditions and facts existent in the intervals between the bouts or seizures?

(1) The mode of appearance and succession of the seizures is not of avail for this purpose. Nor is nocturnal epilepsy specially syphilitic, as it has been alleged to be.

(2) In the second place, as to distinctions to be drawn from symptoms constituting attacks of "syphilitic epilepsy," there is no pathognomonic sign. Some traits may justly arouse suspicion as to a specific origin; such as, in the convulsion of brain syphilis, the usual absence of the initial cry and the much greater frequency of paralysis immediately following the attack. But under this head,

Much more striking, characteristic and useful for diagnostic purposes, are the partial and conscious characters of some convulsive attacks observed in cerebral syphilis. Here (*a*) only a muscular group, or one limb, or one side of the body, may be convulsed; and (*b*) during the convulsion consciousness is more or less retained.

In some cases this retention of consciousness is so marked that the patient becomes a spectator of his own attack—he is said to assist at it—and from it, being conscious, he may suffer severe pain; in other cases the preservation is less complete, consciousness is clouded, and perhaps the patient's replies are monosyllabic only; in still other examples consciousness is confused, obscured, half obliterated during the convulsive movements, of which last the patient does not take much heed or notice, and as to which he evinces no anxiety, at the time.

Sometimes this partial and conscious "epilepsy" forms the first stage of an attack, of which the second stage consists of a more complete seizure with entire loss of consciousness, the convulsive spasms occurring simultaneously, either remaining local and unilateral, or becoming general.

Fournier made far too little of the immediate sequels to these attacks of so-called partial and conscious epilepsy. The mental defects, following, are more frequent and marked than he described; and there is *always* (not merely sometimes, as he allowed) some degree of temporary paresis or paralysis after the attacks.

The partial and conscious character of an "epilepsy" is evidence in favour of, though not proof of, its *symptomatic* nature.

Moreover, the partial "epilepsy" is evidence as to the *seat of the lesions* in which it originates; it is the expression of definite localisations, of lesions affecting the grey substance, or the subjacent white, of the cortical motor zone of the brain.

Therefore, as regards the constituent symptoms of the seizures, "syphilitic epilepsy," in some cases, is distinguishable from the ordinary "essential" form by the attacks of partial convulsion, which go to prove the existence of a *symptomatic* epilepsy.

(3) In the third place, some important diagnostic indications are also to be drawn from the facts relating to the intervals between the convulsive attacks. For, whereas the genuine epileptic, as a rule, is fairly well between the several attacks with their prodromata and post-convulsive phenomena; the subject of syphilitic epilepsy, on the contrary, as a rule, is affected, also, with other orders of cerebral symptoms; such as headache, "congestive" symptoms, sensorial disorders, motor troubles, mental symptoms, aphasia, optic neuritis, &c.

Another important diagnostic element is the mature age at which the first convulsive seizures have occurred.

To resume: there is no pathognomonic sign separating "syphilitic epilepsy" from ordinary epilepsy. Yet in some cases it is distinguished by the characters of "partial epilepsy"; namely, localised or unilateral convulsion with more or less preservation of consciousness during the attack. As a rule, also, it becomes associated, before long, with other cerebral phenomena. And, lastly, it comes on at mature age (in usual examples of acquired syphilis).

But having arrived at the conclusion that a "symptomatic epilepsy" is before us, how do we know that it is symptomatic of specific lesions? This we can only know by (1) a valid history, or indications, of syphilis in the past, or (2) the existence in the present of other clear manifestations of syphilis in various parts of the body; and by (3) the exclusion, diagnostically, of possible effective factors other than syphilis; together with (4) the results of anti-syphilitic treatment.

Sometimes we may not have all of even these few points to help us. For example, called to a patient of whom we know little, our only available guides may be the history of preceding syphilis, and the comparativly late age at which convulsion first occurred.

Recently, Fournier has described "para-syphilitic epilepsy" as maintaining through many years the character of true epilepsy, *i.e.*, not associating itself with other cerebral symptoms; as pertaining to tertiary syphilis, and usually coming from ten to twenty years after infection; as presenting both the major and minor attacks; as uninfluenced by anti-luetic treatment, but improved by bromides; and as probably not conditioned by the recognised specific brain lesions of syphilis, although of syphilitic origin.

B.

In section "B" of this part it is proposed (1) to summarise, and then (2) to comment upon, the recent important article of Prof. Paul Kowalewsky[1] on this subject.

I.

He accepts, and builds his articles upon the basis of, a division of epilepsy into (1) cortical epilepsy, and (2) medullary (or idiopathic) epilepsy. Taking these terms, for the moment, in the sense in which they are used, it is evident that until comparatively recent years the former was included in the description of the latter, was not distinctly separated from it, and, though sometimes described as spasmodic epilepsy, even the description of partial spasmodic epilepsy was not altogether that of "cortical" epilepsy, often now so-called.

"Cortical" epilepsy, so-called, is to a large extent, equivalent to syphilitic epilepsy, especially that of *acquired* syphilis, which readily provokes "cortical" convulsion, and is perhaps the most frequent source thereof. In hereditary syphilis, gummata, &c., may induce "cortical" epilepsy, but in many cases the inheritance is, not of syphilis, at least, not of specific local-lesion-making yphilis, but of a weak, defectively stable and resistive, central ervous system.

The epilepsy may come as a result of syphilitic dyscrasia, rhaps affecting nutritive and circulatory centres.

But it may appear as a partial cortical epilepsy, from a circumscribed cortical lesion or disease. In this latter case the results of anti-luetic treatment may be brilliant, the convulsion may only first appear in middle or later life, and is preceded by an aura in the parts which are left temporarily paralysed after the attack. Sooner or later, symptoms of focal lesion usually co-exist, but in some cases, the centres at fault in originating convulsion are roused to morbid action indirectly, by incitation from a distance.

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[1] *Berliner Klinische Wochenschrift*, Jan. 22, 1894.

Epilepsy in Hereditary Syphilis.

According to Kowalewsky *hereditary* syphilis presents itself as of two kinds. One as degenerated, altered, weakened, metamorphosed syphilis; the syphilitic diathesis, so-called, in which the syphilitic contagium is inherited in its chemical form (so to speak), and so far as convulsion is concerned, its effect usually appears as essential, idiopathic, or medullary epilepsy.

The other presents itself as inherited gummatous lesion, either diffuse or syphilomatous [to which should be added[1] the words—or an actively working virus, tending to produce such] giving rise to cortical epilepsy, usually unilateral, or even very local and limited in range.

Inherited syphilis mostly passes over from parent to fœtus as chemical substance, probably chemically combined (or united) with the other elements of the embryonic cells, or serves in these cells, as an equivalent of another chemical substance. In further embryonic growth, this noxious substance may concentrate strongly in the central nervous system, whereby this last is brought into a pathological state, which may manifest itself in all degrees, from gross naked-eye malformations and developmental defects of the brain, down to chemical and molecular defects and alterations in the brain cells, not demonstrable by our methods of examination: and, clinically, ranging from idiocy to slight neurasthenia; but also affording a basis for the later development of neurosis and psychosis of every kind. Taking up only the slighter forms of change, we see, on the basis of hereditary syphilis, the development of neurasthenia, epilepsy, hysteria, chorea, &c The brain elements containing the noxious substance, seem as impotent and unresistive towards hostile influences; this leavin an open way for the easy development of every morbid ment and neurosal state. There is, in fact, here, not a special syph litic neurosis, but a tendency to nervous disease. Thus, in som cases of hereditary epilepsy, syphilis, like alcohol, acts only as a favouring agency. Here the hereditary syphilitic epilepsy is of the essential, idiopathic, "medullary" kind.

It is possible, says Kowalewsky, that in some such cases the syphilitic contagium poisons those of the nervous elements of the medulla [oblongata ?] which serve as centres and starting point of the epileptic attacks, and that it renders their nerve cells more irritable and less capable of resistance. Here, there is irregular function of nerve cells of epileptic sub-cortical centres, owing to abnormal chemical or molecular structure. Therewith operates the syphilitic contagium, either weakening the usual chemical stability of the nerve cells and calling forth quantitative change, or combining with the cell substance and evoking qualitative alteration.

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[1] See Comment No. 5, below.

On the other hand, "cortical" epilepsy of hereditary syphilis comes from circumscribed or diffuse formations in the meninges or brain cortex, or sub-cortical ganglia, the diffuse being chiefly in the vascular system. Hence "cortical" hereditary syphilitic epilepsy may present two sorts: the one, with circumscribed formations strongly pronounced and with the characters of quite partial cortical epilepsy; the other, more extensive, and often united with delayed development and innate malformations, and on which, again, polio-encephalitis or meningitis may supervene.

Consequently the two great forms of hereditary syphilitic epilepsy are sharply separated by the mechanism of their commencement, by their origin, prognosis, and treatment.

And one of them, namely, "medullary" hereditary syphilitic epilepsy is taken as having its source in paternal syphilis, whilst the other of them, namely, the "cortical" kind, is held to take origin in syphilis of the mother.

Epilepsy from Acquired Brain Syphilis.

Epilepsy from acquired brain syphilis has usually (or always) the "cortical" type, but even so the mechanism of the origin of this "cortical epilepsy" presents varieties.

(1) The "cortical" epilepsy may be set up (*i.e.*, indirectly, W. J. M.), by gumma of meninges or cortex; or,

(2) By a diffuse gummy process, more intensely affecting cortical and pial vessels at some points or places than at others; or,

(3) During the absorption of diffuse gummy syphilitic products during vigorous anti-syphilitic treatment, by reason of local ritation at the part undergoing resorption ; or

(4) By the irritation produced by the scar and contraction of the brain substance, following disappearance of the absorbed gummata.

In all these there is "cortical epilepsy," but in each having a different signification.

In the first, wherein "epilepsy" is produced through the effects of solitary, or of few, gummata (and we may include here, also, the similar condition in hereditary syphilis—W. J. M.), if untreated, the attacks are unchecked; they are followed by temporary paresis, and, later on, paralysis, and even perhaps contractures. Under energetic specific treatment, the epilepsy may disappear finally and for good, but not seldom it relapses, the convulsion, paresis, paralysis or contracture return, and treatment is without avail. The gumma probably has become replaced by cicatricial tissue and by a local drawing together of the surrounding nervous substance, constituting a focus of incitement to convulsion, and one very little amenable to medication.

Polio-myelitis, or meningitis cerebralis, may come in such cases, and from a syphilitic change in the blood vessels.

In syphilitic epilepsy of this variety the gummata may be recent, may merely compress nerve elements, may yield readily to specific treatment, or they may be of long standing and productive of destruction and disappearance of nervous elements, or the scars and relics of gummata, and of their destructive action may alone remain.

The second variety of "cortical epilepsy," in *acquired* syphilis, develops with a gummatous process in the meninges, or brain cortex, which is diffuse, but some parts of the cortex or membranes, more than others, are affected by this diffuse gummy process in the blood vessels. Under appropriate treatment this may clear up, and the epilepsy pass away. This variety belongs to the lightest of those of syphilitic epilepsy, says Kowalewsky and he adds that it is followed by no post-epileptic processes and paralyses. To both of these statements one may take some exception.[1]

The third variety of syphilitic cortical epilepsy, or that which appears during resorption of the products of diffuse gummy syphilis under vigorous treatment, is one in which the convulsive seizures occur a very few times only, and disappear, leaving n[illegible] traces.

To explain this epilepsy is not easy:—

(*a*) Probably auto-intoxication by augmented products [illegible] metamorphosis acts here. One may readily admit the possibili[illegible] of epileptoid attacks brought about by the formation of certain ptomaines and leucomaines in the organism, particularly in persons with defectively resistive central nervous system and a tendency to epilepsy. This form of epilepsy may rightly be termed *toxic;* often the attacks appear as being idiopathic.

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[1] See, also commentary No. 6, below.

(*b*) Besides this, the vessel walls, in diffuse gummy cortical and meningeal processes, such as have existed in this form of "epilepsy," are filled with neoplastic elements; thus their tonus is much altered. Whilst, under energetic treatment, the vessel walls are being rapidly freed from these neoplastic elements, the irritability and contractility of the vessels is very inequable, and they are inclined to contract suddenly and unequally; as, also, to be dilated and paralysed, and much more markedly so than in normal states. Thus may attacks of cortical epilepsy easily be occasioned, until the normal tone of the blood vessels is re-established.

One or other or both conjointly of these conditions "(*a*)" and "(*b*)" may thus induce this form of syphilitic epilepsy, until:—

(1) The removal from the organism of the unwonted products of regressive metamorphosis occurs; or until

(2) The re-acquisition of due tone of the vessel walls restores a normal circulatory balance and blood pressure in the part, and frees the patient from epilepsy.

For commentary on several of these points see Nos. 2, 3, and 4, below.

The fourth variety of "cortical" epilepsy in acquired syphilis, or that from cicatricial changes and contraction following gummatous processes in cortex or meninges, has already been sufficiently dealt with, incidentally, in discussing the first variety.

Medullary (idiopathic) epilepsy in acquired syphilis has not been observed by Kowalewsky himself, and he asserts that it is rare. Here must be reckoned the cases of syphilitic epilepsy occurring during the secondary stage of syphilis. This is in consequence of intense concentration of the syphilitic contagium in the central nervous system, and particularly in the region of the "convulsive centres," says Kowalewsky. But we suggest that robably the toxæmic state, equal everywhere in the blood-mass, s sufficient, the vulnerability of the unstable nervous elements, y it, being the effective relation; and selective affinity, for and by —and not concentration of—the toxic substance, being the real modus.

Whereas this form, in which toxæmia is due to the syphilitic virus or its products (toxines), is termed idiopathic ("medullary"); yet that in which products of regressive metamorphosis of diffuse gummy formations which are rapidly dispersing under treatment, enter the vessels and occasion an analogous condition of toxæmia, is termed "cortical" by Kowalewsky.[1]

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[1] For comment on this, see No. 3, below.

Summary.

Thus, in short, may all cases of syphilitic epilepsy be placed in two great groups—congenital, and acquired.

Congenital syphilitic epilepsy has two forms: the idiopathic ("medullary"), which almost always develops by reason of irresistiveness and change of the central nervous system produced by the syphilitic contagium, and therefore is rightly termed dyscrasic epilepsy: and the cortical called forth by inheritance of an organised gummy process.

Acquired syphilis also manifests itself in the forms of cortical and idiopathic ("medullary"); the former under the action of isolated gummata, irregular gummy processes, and scars at the site of former gummy formations; the latter under the influence of infection of the organism by the syphilitic contagium, and by the products of regressive metamorphosis, under vigorous antiluetic treatment.

II.

Commentary.—This writer carries one so pleasantly along that judgment is soothed to rest as if by a mental opiate; but I think issue must be joined on several points.

(1) To begin with, the division of epilepsy into "cortical" and "medullary" is one to which we do not adhere. By "medullary epilepsy" (which he does not define), we take it the author means epilepsy considered as a condition of which the clinical symptoms are determined by disorder starting from the medulla oblongata, according to the theory supported by Brown-Séquard and others more than thirty years ago. Under this term "medullary epilepsy"; which Kowalewsky and others use as contrasted with "cortical epilepsy"; are included the cases of epilepsy *par excellence*, those which were chiefly concerned in the older descriptions of epilepsy, and as an appanage of which the more recently (or more generally) separated epileptiform seizures were considered, and the description of which former these seizures at one time modified by their inclusion therein.

But surely all the chief epilepsies are cortical, *i.e.*, the cortex is essentially engaged in them. One may admit that disorder of *motor* grey matter, anywhere in the cerebro-spinal nervous system, may occasion convulsion, or at least an analogous local spasm—analogous, *i.e.*, to a convulsion, or amounting to a convulsion of slight extent, or simple lowly type. Is not "idiopathic" ("medullary") epilepsy as much an affair of the brain cortex as is the epileptiform seizure indirectly set up by a meningeal or cortical gumma? Indeed, is not the former *more* of a cortical affair than is the latter, involving; as the former does in *grand mal*, large areas, possibly nearly the whole of the cortex? But be this as it may, we accede to Dr. H. Jackson's view as to the implication of the cortex, and Prof. Horsley's experiments on the simian or canine cortex seem to corroborate it.

If we discard the name "cortical" as being misleading if applied to one group only of epileptic cases, how shall we best express the facts? Shall we confine the term epilepsy to what has been called "essential" or "idiopathic" epilepsy, and call the rest "epileptiform or epileptoid convulsions," or "convulsions" merely; or shall we call the former "epilepsy proper," and the latter "symptomatic epilepsy"? This last might be convenient, but is open to the cavil that all epilepsies are symptomatic; although of what symptomatic we do not always know. Or shall we speak of "functional" and of "organic" epilepsy?

Then again, the symptomatic, or even idiopathic, epilepsy may be aroused by excitation at a distance from the nervous apparatus fundamentally at fault. And an important group of epilepsies (convulsions) is produced by various states of toxæmia, whether these last are engendered by the toxines of infectious disease; by drug and chemical poisons; by noxious substances in food, or the outcome of perverted digestion, altered metabolism, or excretory inadequacy of a positive or of a relative kind.

These last are much more analogous to so-called idiopathic epilepsy, than, *e.g.*, is "epilepsy," so-called, from (*i.e.*, the indirect outcome of) intracranial gumma. And, indeed, it is possible that a recurring toxæmic state, or irregularly periodical cumulative tension, resulting from a form of slight auto-intoxication may be one factor in the production of ordinary idiopathic epilepsy, or of some examples of it. For such special form of auto-intoxication might well be due to a functional innate perversion or defect, inasmuch as even structural congenital peculiarities and deviations are observed in undue degree in many epileptics (as, *e.g.*, the osseous, the cerebral).

In the article under discussion, besides (1) the division into "cortical" and "medullary," several other points have been noticed, incidentally, as affording ground for the expression of a different opinion. Such are the following:—

(2) The self-contradiction between the definition of the third form of cortical epilepsy (p. 79, col. 1), and the description of it in the text (p. 80): *i.e.*, between (*a*) the definition as local

irritation at the site of a rapid absorption of diffuse gummatous products, and (*b*) the description as a toxæmia induced by that rapid disintegration and absorption.

(3) The self-contradiction involved in naming "cortical," and describing under that head, the epilepsy due to absorption of products of rapid regressive metamorphosis of gumma, it being in fact, toxic, and therefore a variety of idiopathic ("medullary") epilepsy according to the nomenclature used in the article itself.

(4) The fact that in the third variety described as of "cortical" epilepsy, "cortical" and "medullary" epilepsy, so-called, intermingle; and thus a cross-division is formed.

(5) The qualification that must be appended to the assertion, made impliedly, that cortical epilepsy, in inherited syphilis, means the inheritance at birth, of ready-formed syphilitic gummy neoplasms, or diffuse formations, in meninges or brain; inasmuch as these may form *after* birth.

(6) The denial, that must be made of the statement that "cortical epilepsy" from a diffuse gummatous process is not followed by any post-epileptic phenomena and paralyses, it being invariably followed by some degree of post-convulsive paralysis or paresis.

C.

We now pass entirely away from the excellent article that has been the occasion of the preceding lines.

In all discussions on epileptiform seizures, and of epilepsy, it is very necessary not to lose sight of the influence of alterations of blood-pressure and blood and lymph circulation in the brain, and the relations of these to the cerebral cell elements.

I knew of a case in an asylum in which a portion of the skull-cap had been lost, the part of the brain underlying that area being covered only by the scalp and the meninges. The irregularly recurring convulsions could always be accurately forecast by the preceding bulge of the scalp over the hole, under the increased pressure, from within, of the intracranial contents.

Incidentally, we have noticed how rare a distinguished observer holds "idiopathic" epilepsy to be in acquired syphilis; such cases, for instance, as have been reported in the secondary stage of syphilis. I think it cannot be so very rare, having seen several examples of it, and published two such.[1] Both were cases of epilepsy coming on during the secondary stage of syphilis. In one, chancre was followed in four to five months by "epileptic" seizure, and this quickly by mania, three months after the onset of which there were amygdaloid inguinal and cervical glands and a mucous patch. In the other, the epileptic attacks preceded insanity by several months (five and seven). At its onset, the insanity was termed, elsewhere, "acute dementia," and there was refusal of food; subsequently there was acute, and later on, chronic, mental excitement. On admission, were a papular rash, large hard inguinal glands, a sallow dingy pallor.

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[1] *Brit. and For. Med. Chir. Review*, Oct., 1876, Case 3 in table, p. 451, and text p. 447, and Case 14 in table, p. 454.

As examples of epileptiform convulsions in intracranial syphilis, may be mentioned cases already published by the present writer, such as one with gummatous inflammation of meninges; invasion and destruction of parts of convolutional surface, partly from blockage of vessels; both diffuse and syphilomatous arteritis, chiefly of larger vessels; secondary softenings. (Necropsy.)

Another, with severe osteocopic cranial pains, pachy-meningitis, and dural gummata. (Necropsy.)

Another with pericranial nodes, and severe osteocopic cranial pain. Diagnosis; basal pachy-meningitis (and meningitis?) chiefly posteriorly, gummatous or simple. (Recovery.)

None of these adults had had convulsions until there were other indications of macroscopic intracranial syphilitic disease.

I think we may divide convulsions (not calling them epilepsies) directly or indirectly of syphilitic causation or origin, into :—

(1) Those arising from specific syphilitic intracranial lesion or morbid process, *i.e.*, cellular hyperplasiæ, diffuse or circumscribed, of inflammatory, or quasi-inflammatory nature, in encephalon, or in meninges, or in arteries, and either of a simple type and tending to sclerous change, or of gummatous type; or those arising from histological sequelæ of these lesions.

(2) Those arising more directly from alterations, of syphilitic origin, in blood circulation and pressure, and in brain pressure.

(3) The toxic, or those from what I term syphilæmia, *i.e.*, syphilitic virus or toxine in the blood in the secondary or later stage; or from toxæmia owing to resorption of the products of rapid katabolism of gummy material.

If it exists a congenital or acquired instability of nervous centres will co-operate with all the above.

(4) Those in persons who have inherited both syphilis and a dyscrasia of syphilitic origin manifested (*inter alia*) in nervous instability.

D.

Finally, a word as to nomenclature.

It does not seem to be scientific to call by the same name "epilepsy," the two very different things; to wit, on the one hand:—

First—The epilepsy proper (as it will be called here), having its close relationships with hysteria, with insanity, and with criminality; being—in persons sprung from a neuropathic stock, and presenting somatic and psychic stigmata of neuropathic degeneration—the outward and visible form of an inward and innate vice of nervous-nutrition—of a great constitutional neurosis—the nervous elements being morbidly unstable, liable to gusty, stormy, quick, mercurial change, culminating in paroxysmal outbursts; persons in whom the epilepsy appears from birth or in early life, or in youth; tends to grow with the growth and strengthen with the strength of its unfortunate victims; and, although often associated with obvious developmental defects and deviations, as in cranial bones and in brain, usually presents no neoplastic or other gross perceptible *primary* encephalic pathological change:—and, on the other hand:—

Second—The convulsions, indirectly occasioned by, let us say, the formation of a syphilitic gumma in the meninges or brain of a person probably healthy, or at least not convulsive, hitherto, although now of mature years; the convulsions coming as an accidental occurrence or episode in physical and mental life, and being traceable in ultimate origin to new organic lesions of a grave kind.

To call these two things by the one name, "epilepsy"—because convulsion is a striking, overt, dramatic feature in both—seems to me, much as if one clubbed together under the same name—and in consequence of the mental defect in each—on the one hand, the imbecile; and, on the other, the adult whose mind fails because a crowbar has been blown through his head and brain.

PART VI.

SYPHILITIC NEURALGIA.

Syphilitic neuralgiæ have recently been the subject of some clinical lectures by Professor Obolensky.[1]

The first case was that of a male, aged 36, with an old syphilitic history; and some remaining lymphatic gland enlarge ment. The pain had first come on nearly five years previously, was worse at night, affected the region of the second, third and fourth intercostal spaces on both sides, was more severe on one side than on the other, and presented the painful points of Valleix at the usual situations.

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[1] *Berliner Klinische Wochenschrift*, Feb. 12 and 19, and April 16, 1894.

That the skin is not the part affected is shown by the normal condition of all forms of its sensibility; nor does rough manipulation of the skin increase the pain.

Nor is the muscular tissue affected, because no rheumatic condition, or causative factor of such, exists; and the pain; instead of increasing directly as the pressure increases, as is the case in rheumatism; is increased by slight or moderate pressure, mitigated by strong pressure.

The painful points and their situation, the increase of pain under slight pressure, with its mitigation under strong pressure, the paroxysmal occurrence of the pain, which in the interval remits or ceases—indicate intercostal neuralgia.

The signs of pleurisy are absent.

Neuritis has not existed, for there is no reaction of degeneration on electrical examination, no wasting of muscles supplied by the affected nerves, and no disorder of cutaneous sensibility.

Neuralgia may be produced, as a secondary disease, by mechanical pressure or irritation, as, for example, by tumour; the cause arousing it may be in brain, cord, meninges, or in an inflammatory or other affection of some neighbouring organ or part; or the neuralgia may be reflexly roused from or by, some morbid distant part or organ; or be rheumatic; or follow general nutritive disorder, as, *e.g.*, in the dyscrasia of malaria or of syphilis; or in disorders of hæmato-poietic organs, *e.g.*, chlorosis or leukæmia; or in hysteria, neurasthenia, &c.

Excluding most of the above, for this case, as not supported by examination of the patient, the question whether the neuralgia is malarial comes to the front. With regard to this, besides the general features of neuralgia, above mentioned, the malarial forms present attacks at different times by day or night, and are not fixed, not limited to night time; feelings of chills and heats, and slight rises of body temperature, are apt to occur; intermittent fever has, perhaps, preceded, the patient lives, or has lived in an aguish district. Anti-malarial treatment (quinine and arsenic) assists diagnosis and cures. So, also, does a change of home to a non-malarial district.

Syphilitic neuralgia is distinguished by :—

(1) The history of syphilis.

(2) The existence of traces of past, or evidences of present, syphilis.

(3) Increase of pain at night, its occurrence in paroxysmal attacks at night, the osteocopic character of the pain.

(4) The bilateral disposition of the pain, other forms of neuralgia being (usually) unilateral.

(5) The absence of electrical reaction of degeneration, not-

withstanding the protracted duration of the neuralgia (years perhaps).

(6) The apparently groundless appearance of the pain.

(7) The absence of other causative factors.

Treatment.—By subcutaneous injections of calomel (two only, and at a week's interval, and of about 2 grains each); thermo-cautery over the affected parts; hot baths; galvanisation.

Result.—Recovery, and attributed to the calomel injections.

The second case is that of a man, aged 43 years, who has neuralgic pains in the seven upper intercostal spaces on both sides, but worse on one side than on the other. The sensibility of skin and the muscles are normal. There is no reaction of degeneration, no pleurisy, or vertebral or costal disease. The patient is anæmic, of ill-developed lax musculature. Thickenings, tender on pressure, exist on the frontal eminences and over the nuchal bones, and are painful at night. At the junction of the first and second ribs with the sternum are similar thickenings, tender on pressure.

Left cardiac hypertrophy, double aortic murmur, and aneurysm of the aortic arch are present, and the radial and temporal arteries are sclerosed, harder and more rigid than normal. But the aneurysm is not so situated as to be able to occasion the neuralgia present. Aneurysm of the aortic arch may give rise to a form of neuralgia, or angina pectoris; aneurysm of the descending thoracic aorta may, by pressure, cause intercostal neuralgia, and weakening and retardation of the pulse in the abdominal aorta. Usually, the neuralgia with aortic aneurysm is unilateral.

The patient denies syphilis. Twenty years married, he has five children, whom he states to be healthy, but his wife has twice aborted, and six children have died in infancy of "spasms."

But the diagnosis of syphilis rests on the bone conditions, and tenderness, swelling and pain, already mentioned, including their particular site; on the nocturnal, paroxysmal character of the nerve-pain in head, chest and legs; the enlargement of lymphatic glands in nuchal, axillary, cubital, and inguinal regions; the nocturnal osteocopic leg-pains without periostitis; the long duration; the "groundless" bilateral neuralgia; and the absence of reaction of degeneration.

To syphilis, also, he inclined to attribute the disease of aortic arch and aortic valves, the insufficiency of which explained the vertigo, dyspnœa, and aortic pulsation.

Treatment.—Calomel hypodermically, anti-neuralgic galvanisation, liq. arsenicalis, tr. gelsem. sempervir.

Result.—Rapid recovery from the neuralgia.

The third case, that of a male, aged 43 years, began eighteen years ago with pains in the region of the left kidney, radiating to the scrobiculus cordis and to the breech. The pains began without apparent cause, at first were not very severe, came on only at intervals of about a month or more, and lasted from twenty to thirty minutes. In the course of several years, they gradually increased in frequency, duration, severity, and extent; so that at last they came on every night, remained for about eleven hours, and, except during short remissions, were intolerably severe; they also took a wider range over left of abdomen and to shoulder and scapula. They ceased suddenly each morning. The night urine, passed during the attacks of pain, was from three to four times the amount of the day urine; it was acid, very pale, its sp. gr. 1008; it was free from abnormal constituents. Insomnia, of course, occurred at night during these pains.

The inguinal glands are enlarged and hard, and those of the neck and axillæ are enlarged. He is married, and has three children, they, and his wife, who has aborted twice, are healthy. At the age of 20, he incurred a hard chancre, followed by enlargement of lymphatic glands and a rash. (*Hg.*)

Vomiting, &c., often occur at the close of, or during, the attacks.

Is the case one of renal colic? Against this diagnosis, are the absence of ureteral pains, or of pains radiating into testicle, penis, or thigh; the absence of any hæmaturia, or of the passage of any calculi or gravel, during the long space of time presenting so many and such severe attacks of pain; the absence of pain in, or tenderness on pressure over, the kidney, and of abnormal urinary deposits. Movement, straining exertion, or an incommodious vehicle, do *not* affect or bring on the pains, as they are apt to do with renal calculus; and, unlike what occurs in this last, the pain is straitly confined to the night-time.

The long duration of the shooting pains, without any other indications as of tabes, shows that the attacks are not the renal crises of that disease.

Nor is the case one of diabetes insipidus; the over-secretion during the night of pain is fairly well balanced by the under-secretion during the day of freedom; and there is no thirst There are no symptoms of interstitial nephritis.

The pains are neuralgic, coming on without apparent cause, and ceasing suddenly, sometimes with vomiting.

The pains are partly in the same places as those of renal colic, and during them the function of the kidneys is disordered. Therefore the source of the neuralgia appears to be the nerves which innervate the kidneys, and both the sensory and those which regulate the vascular tonus are affected. There is neuralgia of the splanchnic nerve, accounting for the pain; and, on the vasomotor side, dilatation of the renal blood vessels and increased watery secretion of urine, owing to periodical changes in the splanchnic nerve, or, perhaps, renal plexus.

The neuralgia is not symptomatic of a cerebral, or spinal, or bony disease. Nor is there the slightest evidence that it is reflexly-produced pain; nor is there any indication of neuritis of ordinary full-blown type.

That the origin of the neuralgia is not malarial is concluded on grounds similar to those for the same conclusion in a case stated in summary above. That the neuralgia is of syphilitic origin is concluded from the nocturnal character of the pain, the history of lues, and the enlarged lympathic glands.

Rapid recovery under (as he refused Hg.) K.I., Na.Br. Gelsem. Semp., As., thermo-cautery over spine, galvanisation.

(K. I. fails in some cases.)

The diagnosis of syphilitic neuralgia rests on:—

(1) The nocturnal occurrence of the neuralgic attacks.

(2) If intercostal the neuralgia is bilateral.

(3) The absence of the reaction of degeneration and atrophy, notwithstanding the long duration of the neuralgia; such signs indicating neuritis rather than neuralgia.

(4) The appearance of the pain without apparent occasion.

(5) The history or indications of past or present syphilis.

In contrast with the above, may be mentioned two cases of irritative peripheral syphilitic neuritis reported by S. Ehrmann [1]

One was a coachman, aged 38. Paræsthesia in region supplied by the left ulnar nerve occurred eight and a-half months after syphilitic infection. The interossei and other small hand-muscles were extremely atrophic. Hyperæsthesia affected the areas of distribution of certain nerve-branches in forearm and hand. Galvanic irritability lessened; no reaction of degeneration. The ulnar nerve was somewhat thickened, and in its entire length was painful on contact. Anti-luetic treatment succeeded.

The other was a merchant, aged 32. Pains in left thigh a year after infection. The peroneal nerve distinctly thickened, tender, painful. Hyperæsthesia in the district of the crural nerve; hyperalgesia in the cutaneous areas supplied by the peroneal nerve. Slight bladder and rectum disorders. Vastus muscles very much atrophied.

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[1] *Wiener Med. Wochenschrift*, No. 33, 1893.

Critical Digest.

SOME RECENT PSYCHOPHYSICAL LITERATURE.

Professor Kuelpe[1] has given us a text-book of experimental psychology; and Professor Sanford[2] is writing a Laboratory Course. M. Binet[3] now steps into the field with a popular Introduction to the science, in the compilation of which he has been assisted by MM. Philippe, Courtier and v. Henri. If the first two of these works are good—so good that psychologists may be excused for feeling a little self-congratulatory for the moment—it must be confessed that the last is somewhat disappointing. Opening with a full account of the Laboratory of the Sorbonne, followed by a sketch of the Leipsic Institute, it devotes seven brief and incomplete chapters to the discussion of psychological method and achievement. Two only of these appear to have been written with any degree of enthusiasm—those on movement and on memory. The former, indeed, is so tantalisingly interesting and lucid, that one cannot but wish there were more of it, and more like it. After these rank the sections on "psychometry" (the word looks better in French than in any other language, but is bad enough even there), and on observational method. The remaining three chapters are entitled psychological method, sensation perception and attention, and ideation.

Besides being unworthy of an author of M. Binet's power and experience, the volume sins in the matter of comparisons which are odious. The writers do not state that experimental psychology is *une science française*, but they never tire of congratulating themselves that they are not as the *expérimentateurs étrangers.* This is certainly to be deprecated in the interest of the science: and the danger of a recoil is great—as is curiously illustrated in the present instance. For the great difference between the old psychology (of which Wolff may stand as typical), and the new (of which Wundt was the effective founder) is this, that the former was merely descriptive, while the latter is descriptive and explanatory. The Wolffians—I use the term more broadly than is, perhaps, legitimate—depicted isolated mental facts; described separate complex processes, such as emotion; and furnished none but metaphysical explanations of the course of psychical events. The moderns seek to give psychological theories, that is, to state the psychological conditions of the processes of consciousness; they work not upon a "faculty" here and another there, but upon the total extent of mind, searching for uniformities and principles of correlation. The result is, that the stricter experimentalists—those who define "experiment" in psychology as they would in physiology—tend to work predominantly upon the normal, healthy consciousness, having recourse to the abnormal only for verification or illustration. But it was a result of equal inevitableness for the descriptive school that it should have a keen relish for the abnormal—that it should devote the time it did to the investigation of "genius," phenomenal memories, and so forth. And lo! I have before me, for notice, three other books of M. Binet's, and they are entitled "Les altérations de la personnalité,"[1] "Psychologie des grands calculateurs et joueurs d'échecs,"[2] and "On Double Consciousness."[3] Here is a glass house, in very truth.

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[1] "Grundriss der psychologie, auf experimenteller Grundlage dargestellt," Leipzig, 1898.

[2] "A Course in Experimental psychology," advanced sheets, Boston, Mass., 1894.

[3] "Introduction à la psychologie expérimentale," Paris, 1894.

Of course, I do not mean that the methods of this author are the methods of the seventeenth century. His problems are all conceived in the spirit of modern science. Nor is it anything more than an accident that the volumes under review deal with abnormalities. M. Binet and his pupils have published too many experiments upon memory, tactile localisation, &c., for there to be any valid ground for the counter-criticism that the study of the unhealthy or the out-of-the-way is characteristically French, or that there is any radical difference between the methodology of experimental psychology in France and in Germany and America.

As regards the general question, however, there can be no doubt, as Kuelpe says, that the students of mental pathology are far more indebted to the students of mental physiology than are these last to the former. The very phrase "double consciousness" is indicative. It presupposes, obviously, that in employing the term "consciousness" we are speaking in terms of the known; by reducing the given problem to a "conscious" formulation, we are helping matters on towards its solution. In other words, the pathologist is appealing to physiology for explanatory analogies. Unfortunately the appeal has an *ad captandum* ring. The phrase seems to have gained an established place in psychological nomenclature; it has been brought into vogue by such men as Taine, Janet, Dessoir, Moll. And yet, as Wundt has pointed out, it is nothing else than a rehabilitation of the old beliefs in demoniacal possession, in second sight, in supernatural illumination. Of necessity, its connotation has been modernised; the "second consciousness" has fallen from its high estate; from being the more important—a special endowment or affliction—it has become the less important, and we talk, in would-be scientific terms, of the "subliminal" mind, or, with Myers of by-paths, stray tendencies of conscious evolution. Are we any the nearer to a real explanation of the phenomena?

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[1] Paris, 1892. [2] Paris, 1894. [3] Chicago, Ill., 1894 (Trs.)

This is all very well, it may be said: but has the "strictly scientific" school any "real" explanation to offer in place of the one it condemns? Now such a question cannot but put the questioned at a disadvantage. For plainly, people can understand and remember the word "phlogiston," but the proof that heat is a mode of motion requires time and patience for its assimilation. So the "hypothesis" of a complex self is readily grasped, and has something fascinatingly uncanny about it; whereas the grasping of the other type of theory means a fairly detailed study of certain portions (not the most easy) of physiology and psychology. If the reader will take the trouble to work through the writings of Moll,[1] Bernheim[2] and Wundt,[3] he will have some idea of the actual difficulty of the problem, and of the reasonings put forward towards its solution. As regards the essay more especially under discussion, it is noteworthy that even M. Courtier[4] will not go the whole way with M. Binet. But where is the line to be drawn?

The volume mentioned above in the second place is more valuable. It contains exhaustive studies of the mentality of MM. Inaudi—a professional "calculator" of the exclusively auditory type—and Diamandi, a visual calculator. It also publishes the results of an *enquête* upon blindfold chessplaying, the outcome of which is, that the players possess an unusual power of sustained concentration of the attention, a highly developed faculty of visualisation, and a reliable recapitulatory memory. Both sets of facts are interesting: they are material upon which the psychophysicist of James' "unborable" category can work by way of further analysis.

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[1] "Der Hypnotismus," Berlin, 1890, second edition.

[2] "Die Suggestion une ihre Heilwirkung" (Trs.), Leipzig und Wien, 1888.

[3] "Hypnotismus und Suggestion," Philosophische Studien, 1892. Also separately.

[4] *The Psychological Review*, i., p. 194.

The third work—a volume in the International Scientific Series, in which the "Animal Magnetism" of the writer and M. Féré had already appeared—contains a popular exposition of the facts of changed personality; dealing with "successive personalities," "coexistent personalities," and "alterations in personality induced by suggestion." It is the author's purpose to avoid theory: "je retiendrai seulement les expériences qui se répètent entre toutes les mains, et qui donnent toujours la même conclusion, quelle que soit la fin cherchée." He has been conscientious in adherence to this plan; and although his own views appear clearly enough when looked for, and the hypothesis of divided consciousness is at times strained to breaking, we have in the book an assemblage of scattered observations which it was well worth while to bring together, and which are stated fully and without prejudice. The title and to some extent the contents of the treatise naturally recall M. Ribot's "Diseases of Personality."[1]

Before taking leave of M. Binet I may, perhaps, call attention to the second edition of his "Études de psychologie expérimentale."[2] The volume contains four papers: on fetishism in love, a pathological study based on the work of Charcot and Magnan but embodying much new material; on the psychic life of micro-organisms; on the intensity of mental images; and on the hypnotic problem, together with a note on hysteric writing. The second article presents a number of valuable biological observations, illustrated by diagrams for the most part after Professor Balbiani. As regards the psychology, that is as it must be, analogical and speculative. The author, though he ascribes to the Infusoria a faculty of choice, is not carried to extremes by falling into the language fallacy, as, *e.g.*, Verworn[3] has been. This "choice," for instance, is "un des faits les plus obscurs. . . . quant à l'interpréter, nous en sommes incapables." But is not the term, on that account, a dangerous one to employ? The third is a discussion based upon hypnotic results, but contains remarks on the relation of logic to psychology, of the association of ideas to belief, which bring the author into closer connection with experimental psychology in the German sense. Lastly, the fourth paper tells us in succinct terms of the gravamen of the Nancy school against that of the Saltpêtrière.

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[1] Chicago, Ill., 1894 (Trs.); Paris, 1889, 3rd ed.

[2] "Le fétichisme dans l'amour, &c.," Paris, 1891.

[3] "Psychophysiologische Protistenstudien," 1889.

Next upon my list stand two volumes dealing with memory.[1] Neither of them can at all compare in importance with the work of Ebbinghaus,[2] or with that of Mueller and Schumann[3] which arose out of it. Professor van Biervliet's study seems to have been intended as a brief popular introduction to psychology in general, and as such it may serve a good purpose. It has hardly anything of the nature of a true monograph. The author explains the fact of retention by a combination of the Maudsley-Ribot-Delbœuf and Wundt theories, coining the word *trace-disposition* to express his view. He finds three kinds of memory: that of fixation—the more frequent the stimulus, the readier the functioning in response to it; that of reproduction, which is regarded as entirely passive, and as governed by the association "laws" of contiguity and succession; and that of identification, a "faculté de l'âme," not (like the other two) common to mind and matter. There follow short paragraphs on the physiology, pathology and hygiene of memory. But the book, comparatively superficial as it is, will at least be helpful to the beginner; which is more than can be said for that of M. Verneuil. This is a rambling, anecdotal treatise, unchaptered and unindexed, dealing with such questions as the reality of innate ideas, physiognomy, the nature of the mind, cerebral retention, inattention as a cause of weakness of memory, "corps éthéréens," the relation of imagination to memory, memory in animals, heredity, a universal natural science, &c. What the author exactly means by his references to the "cells in the grey substance of the immense brain of God" I have not been able to discover. His general drift seems to be —though I may have misunderstood him—that the functions of memory depend upon nervous functionings, and that this dependency need imply neither Materialism nor Spiritualism.

Feeling and emotion have been very prominently upon the psychological carpet of late. The James-Lange theory is under discussion in the technical journals; experimental investigations into the uniformities of the sense feelings have been undertaken in more than one laboratory; and the publication of Lehmann's "Hauptgesetze des menschlichen Gefuehlslebens."[1] marked a distinct advance in affective psychology. Four works call for notice in the present connection: and firstly Féré's "La pathologie des émotions,"[2] and Bourdon's "L'expression des émotions et des tendances dans le language."[3] The former is a ponderous tome of 600 pages, *de omnibus rebus et quibusdam aliis*, written with no petty sense of restriction to the subject indicated in its title. As we are to deal with man, the author seems to say, let us put together everything that we know of him that can possibly be of any use to us. Let us see how he is influenced by atmosphere and temperature, by sounds and sunlight; and mark the effects of physical exercise or the lack of it. We shall get to the emotions in due time. As we do, at about the 170th page.

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[1] "La mémoire," J. J. van Biervliet. Gand & Paris, 1893. "La mémoire au point de vue physiologique, psychologique et anatomique," H. Verneuil, Paris, 1888.

[2] "Das Gedaechtniss," Leipzig, 1885.

[3] "Experimentelle Beiträge zur Untersuchung des Gedaechtnisses," in the *Zeitschrift für Psychologie, &c.*, 1893. Also separately.

M. Féré does not appear to have any psychological theory of the emotion. Whether it is an ultimate psychical process, or a fusion of the pleasure-pain element with ideational masses, or a complex of organic sensations, he does not tell us. His chief principle of classification is that of the sthenic (*vives, excitantes, violentes, intenses*) and asthenic (*dépressives, penibles*) types. But this is crossed by the joy-sorrow division (*agréables, tristes*), by a division based upon cause (*provoquées, suggerées*), and by yet another based upon the objective reference of the emotional state (*morales, maternelles*). The reader who comes to the work for results of psychological analysis, therefore, will lay it down disappointed.

Not that the book does not contain matter enough. It treats, in an encyclopædic way, of the pathological and curative effects of emotion, of the results of excitement and fatigue, of various psychopathic manifestations, of the forms and conditions of morbid emotivity, and of its diagnosis, treatment and prophylaxy. An interesting chapter, which has come into the book for no particular reason that is apparent, deals with the physical signs of hallucination. The pages devoted to attention bring together a number of facts which are often overlooked in the normal psychologies. But it is curious to read such a statement as this, in face of the general pyschological consensus that attentional phenomena are primarily inhibitory in nature:—"L'exaltation générale de l'activité motrice . . . semble demontrer que c'est à tort que l'on fait intervenir l'hypothèse de l'action de l'arrêt, de l'inhibition, dans la physiologie de l'attention." And it is also curious to find attention figuring in a catalogue of emotions (pp. 115, 207, 208); although many writers emphasise the characteristic feeling which accompanies attention; and it is not unusual to have expectation (expectant attention) classed as an emotion in the strict sense.

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[1] Leipzig, 1892. [2] Paris, 1892. [3] Paris, 1892.

The fact that the volume is so altogether unreviewable tempts one to linger upon its by-subjects. A good deal is said about reaction times, dynamography, ergography and the relation of sensation to movement in general, a favourite topic, of course, with the author.[1] I notice that he takes the side of Wallace in regard to the sexual selection question: "Ces mouvements (des oiseaux mâles) ne sont que des décharges involuntaires du systeme nerveux arrivé a un degré extrême de tension" (p. 213). He has a novel theory of the action of the pneumogastric in syncope and death consequent on emotion. As regards the "seat" of emotion, "Le grand sympathique parait être son organe peripherique," "L'emotion est essentiellement un phenomene reflexe generalisé, dont la voie centrifuge est principalement le systeme grand sympathique." Any kind of cortical localisation is impossible. Emotional expression is mediated "par des mouvements des organes dont l'innervation est le plus souvent en jeu, et en particulier les organes circulatoires." Secondary factors are the length of a nerve, its volume, and the vascularity of the regions it traverses. If we compare this mechanical view with the explanations given by Darwin and Wundt we see that it partly coincides with the first (association of serviceable habits) and third (action due to the constitution of the nervous system) principles of the former author, and also with the first (direct modification of innervation) and third (connection of movement with sensation) of the latter. It seems too simple to cover all the facts.

Given a large assortment of newspaper cuttings and of records of cases from medical practice, a good index to medical periodical literature, and a copious stock of patience, and it would be possible for anyone of ordinary intelligence to fill 600 octavo pages on the variety of topics that M. Féré covers. But the result would not be M. Féré's book. The author writes with a kind of suppressed enthusiasm, a settled conviction that all his by-the-ways and first-of-alls will of necessity interest the reader; he has humanity on the dissecting table, and means to know all about it. It is only fair to say, too, that he is very careful in his use of authorities. When once one has learned one's way about the book, it is valuable on many counts.

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[1] *Cf.* his "Sensation et mouvement," Paris, 1887.

I will just mention here, in passing, a much more typically French work—better in style and better in arrangement—upon a special pathological subject, "La contagion du meurtre," by M. Aubry.[1]

M. Bourdon's book brings psychophysics into relation with philology. Emotion is, for him, "un fonctionnement anormal passager d'un organisme animal": a statement which reminds the reader of Ribot's characterisation of attention. It is the mental resultant or correlate of a total "organic" state; and as such, is no more to be reckoned to sensibility than it is to volition.[2] This view is paralleled by the author with Professor James' theory. But it plainly differs a good deal from that, for it is sensations ("organic" sensations) that Professor James insists on, both in his original exposition[3] and in his recent *apologia* in the *Psychological Review*.[4] The word "organic" is, of course, equivocal. In type, however, the two conceptions are similar. A "tendency" for M. Bourdon is "l'habitude d'une sensation." Both terms, then, are used in wide senses, such psychoses as hunger, sex-instincts, the reaction upon a colour, being regarded as emotions, along with anger, fear, &c.

The list of the special laws of emotion is incomplete, but suggestive. It should be compared with Lehmann's sections on the same subject. The criticism of the explanations of emotional expression given by Darwin and Wundt is very acute, and must, I think, be taken account of by future psychologies. I cannot well summarise the writer on either point; the questions at issue are questions of detail.

We now come to the main topic of the work—the spoken word. This is considered under the headings of pitch, intensity, form or quality, duration, pause (*arrêt ou silence*); and then each of these elements is dealt with in composition, successions of intensities, &c., successions of words, syllables, &c., cohesion and dissociation. A final section on special problems treats of the useless elements of spoken language, of linguistic categories, of poetry and of writing.

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[1] Second edition, Paris, 1892.

[2] Pp. 8, 17. Cf., however, p. 9: "l'émotion, comme la sensation, dont elle n'est qu'un accroissement. . . "

[3] "Principles of Psychology," ii., pp. 449, 450.

[4] Vol. i., No. 3.

These chapters are very far from being free of error in philological detail.[1] Such error, however, might be at least partially condoned in presence of a sound methodology. The author's method is the statistical. He analyses laboriously into their elements passages selected under the guidance of certain principles from the works of various authors in different languages. But the method does not lead in his hands to the right *kind* of result. Thus, to take what is perhaps the most striking instance: there are long tables showing the relative frequency of dentals, labials and gutturals in French, German, English, Italian, Spanish, Celtic, Russian, and Hungarian. But these languages, instead of evincing differences of such a sort that they might be correlated with emotional differences, give all of them about the same proportions of usage. The writer, instead of modifying his method, proceeds irrelevantly to evaluate this result, deducing from it (1) a general law of human speech—general, because of the eight languages one is not Indo-European; and (2) referring it to the great tactile sensibility of the tip of the tongue, its motility, and its juxtaposition to the teeth. It need hardly be remarked that the generalisation is over-hasty; and that, even if true, it is not the kind of generalisation wanted in the book.

The author summarises his work in three propositions. (1) The only direct action of emotional intensity upon ordinary speech is dynamogenic. Moderate emotion increases the force of respiration, and with it the intensity of the spoken word; violent emotion weakens or inhibits the voice. (2) Language is primarily a phenomenon of imitation. (3) The degree of organisation and of stability of linguistic combinations (*unités*) is, other things equal, inversely proportional to the number of elementary phenomena included in them. Not only does M. Bourdon here do his chapters injustice; he has again, it would appear, lost the point of them. Is the influence of emotional intensity, *e.g.*, permanently embodied in the utterance of particular peoples? We are not told.

One lays the volume down with an irritated wish that, containing as it does so many indications of ability, it should not be altogether better. But it is, in any case, well worth reading.

I pass to M. Godfernaux' "Feeling and Thought."[2] The former term covers the phenomena of the affective life, the origin of which is subjective or internal; the latter those of the representative, whose origin is external or objective. The aim of the work is to reinstate the first in their proper place in the structure of mind, in the place from which they have been deposed by current associationist theories.

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[1] This has been pointed out by the anonymous reviewer in *Mind*, new series, vol. ii., p. 403.

[2] "Le Sentiment et la Pensée," *Thèse de Doctorat*, Paris, 1894.

The author sets out to find the two psychoses in isolation. He discovers a morbid exaggeration of thought in mania and hypochondria. "Dans la manie, la connaissance du monde extérieur, dans l'hypocondrie, les éléments de la notion de notre moi corporel sont peu à peu dissociés jusqu'à la sensation ou l'image simple." In melancholy and ecstacy, on the other hand, feeling is seen in all stages of exaggeration, up to absolute predominance. Finally, in chronic delirium the quantitative relation gives place to a qualitative; we have "une succession définie et constante d'états affectifs differents, correspondant à des modifications definies et constantes de l'association des idées." I do not know how far the correlation of hypochondria with mania will meet with medical approval; the writer's analysis is ingenious. But, that apart, he evidently pushes his instances beyond what they will bear, in his eagerness to exemplify his standpoint.

Passing to the study of the normal consciousness, M. Godfernaux notes the occurrence of affective states of excitement and depression similar in kind to those observed in the abnormal mentality. He further proceeds to show that the affective state is normally *l'agent profond* of the association of ideas. In order to make his view of the interrelation of the two sides of mind more precise, he has recourse to the phenomena of movement, and concludes that "the work by which the vague and indefinite effort of the tendency (*tendance*) becomes definitised in more or less complex groups of motor phenomena, and, ultimately, in very well defined muscular co-ordinations, shows a constant correspondence to the work by which the emotion takes on a concrete and definite form, and creates a definite synthesis of the elements of consciousness, sensations or simple images." Four appendices deal with the evolution and classification of mental derangement, and a fifth records some observations on chronic delirium.

The writer might with advantage have been more exact in his use of terms. "Feeling," he describes as "un phenomène de conscience diffus et vague." It apparently includes affection proper and rudimentary conation. Had the literature been better searched, this vagueness could have been removed. All citations (except one from Horwicz and one from Lourie) are from French works or translations; thus, Wundt is quoted from the French version of the second edition; Lehmann is not mentioned, although he and M. Godfernaux treat in part of the same topics. At the same time, the book is welcome as indicating a reaction against the exclusively associationist position. The recent articles upon attention and apperception in BRAIN will have made its readers familiar with the fact that there are a large number of psychologists who cannot rest content with a reduction of conscious life to sensational terms. Emotion is, like attention, a process requiring other conscious elements for its satisfactory explanation.

Dr. Irons has been writing in *Mind*[1] recently to show that the emotion is not a complex of elementary mental processes, but itself a fact of mind which is *sui generis*. The ordinary opinion is that emotion is a compound of the affective element (pleasure-pain) with ideational masses, these latter differing in the different emotions. Just as a headache and a toothache differ only sensationally, the sensational substrate of the affection being different and enabling us to localise the aches in forehead and tooth, and are identical in affective quality, the same *qua* pains, *qua* unpleasantnesses, so the emotions are all alike pleasant or painful, all fall within one of these two affective qualities, and differ simply in regard to the trains of ideas, associations, imaginings, &c., which constitute them on the intellectual side. This view does not, of course, as so many authors assert it does, attempt to reduce all concrete pleasures and pains to one type, and all the various emotions to simple pleasure and pain. It says merely, that just as the musical chord has a quality of its own (the piano chord being, *e.g.*, readily distinguishable from the violin clang), but yet is composed in the last resort of a number of simultaneous separate sensations, so the various emotions, qualitatively different as they are in immediate experience, admit of ultimate analysis into one of the two affective qualities, *plus* a particular set of ideas. That is why the emotions cannot be classified; to classify them one would have to classify one's ideas, for any idea can enter into an emotion.

I have dwelt upon this point because M. Danville's little volume on Love[2] is intended to prove that this emotion is irreducible to any mental elements beyond itself. After a somewhat tedious introduction, and criticisms of the views of literature and philosophy (Bain, Spencer, Mantegazza, von Hartmann, Schopenhauer, Delbœuf, Beaunis, Pierre Janet, Féré, de Fleury), we come upon the author's definition. Love is a specific emotive entity, consisting in a more or less permanent variation of the affective and mental state of a subject, occasioned by the realisation (due to the fortuitous initiation of a specialised mental process) of an exclusive and conscious systematisation of his sexual instinct upon an individual of the opposite sex. The phenomenon is, as a rule, accompanied by an exaltation of desire. The first clause means that love "est irreductible à tout autre sentiment." I do not suppose that anybody would dispute this. The exaggeration of desire is regarded as a purely secondary, indirect character. The specialised mental process which "préside à l'origine de la passion amoureuse" is treated in a special chapter, entitled "Psychological Theory" of love. Life at first is asexual. Then follow the various stages of sexuality; unselective sexuality, selective sexuality, relatively systematised selection, love. Each of these has correlated with it a special level of intellectual development. The particular intellectual process which initiates love is a conscious perception of what has heretofore been an "unconscious perception," the perception of an image representing the synthetic type of the particular preferences of the individual from the point of view of sex.

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[1] New series, vol. iii., pp. 77, ff.

[2] "La Psychologie de l'Amour." Paris, 1894.

A second point discussed in the book is that of the nature of love as normal or pathological. The writer sums up the evidence, as he has done before in shorter compass in the *Revue Philosophique*,[1] and decides for normality. "Il est un argument décisif, criterium de l'utilité, qui demontre que l'opinion [contraire] ne parait pas soutenable."

It is clear that there are weak points in the definition which forms the pivot of the whole discussion, even if the author had not incidentally committed himself to the phrase "une passion aussi complexe que l'est la passion amoureuse." But apart from this the chief interest of the book is that it is a book of tendency. It is popularly written, and makes no claim to furnish anything more than a cursory treatment of its subject.

"Die drei Wege des Denkens,"[2] by Dr. I. Paul, is an essay upon the three possible modes of thought about "things"—the physical, psychological, and metaphysical. The writer is influenced almost exclusively by Schopenhauer and Fechner. He insists very strongly upon the fallacy involved in the concept of "transcendent causality"; the doctrine that the interrelation of mind and body is to be conceived of as causal.

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[1] March, 1893.

[2] Leipzig, 1891.

Professor Fano's lecture on the "Physiological Substrate of Thought"[1] is interesting and suggestive. After stating the parallelistic principle in a simple form, he gives his auditors, again very simply, the key to the structure of the nervous system, and gets to his special topic by way of some experiments on *Emys europœa*, made by Francesco Redi in the middle of the seventeenth century. He goes on to present a functional analysis of the tortoise brain, using it to illustrate the fact that all katabolism is normally followed or attended by anabolism. "Ma perchè gli elementi che constituiscono l'organismo continuino a vivere ed a funzionare, occorre che al lavorio distruttivo della molecula organica, che è il sottostrato chimico delle nostre funzioni di movimento, corrisponda un' attività riparatrice per la quale si accumulino di nuovo nella cellula funzionante quelle energie potenziali che essa ha speso col lavoro." Consciousness (will and perception) is correlated with the first phase of this double process; it is a phenomenon of inhibition. "Come le resistenze interposte in un circuito elettrico possono esprimersi sotto forma di luce e di calore, così la coscienza che accompagna i movimenti provocati dal cervello e la intelligenza che per essi si rivela, sono il risultato della resistenza presentata dal cervello medio nell' ambito psichico." Its second phase, of organic synthesis, constitutes the physical basis of memory, "il fondamento delle funzioni mnemoniche." There is nothing new in these views, of course; but the lecturer puts them well. Original with the writer, on the other hand, is apparently the following opinion: "Mi sembra si possa concludere che gli stimoli alle attività trofiche di ricostituzione organica consistano in alcuni casi almeno nei prodotti, nelle scorie, nelle ceneri che sono il risultato delle funzioni."

Still lighter in matter and treatment is the same author's "Le funzioni del cuore nei sentimenti."[2] The paper opens with a sketch of the mechanics of heart-beat and a reference to the part played in physiology by rhythmically recurrent processes. It then passes to psychophysics; the surface of the heart is insensitive, the movements of the organ beyond our volitional control. Why, then, do language and popular opinion so universally regard it as the seat of feeling, passion, and emotion? Because of its unique connection with the brain. This connection is such that, in the first place, the heart is the principal bodily reflector of the mind: "Quasi ogni stimolo che arrivi al cervello dall' esterno, ogni pensiero che vi si svolga, ogni emozione che lo scuota può essere trasmessa per la via dei nervi cardiaci al cuore ed esprimersi nei suoi palpiti." Heart is to brain as actor to dramatic author. In the second place, we have the heart reflecting physiologically caused emotions; it may be by way of the nervous system again, or it may be under the direct influence of the quality of the blood. The tissues are composed of numberless minute organisms, cells: "Il cuore per le sue disposizioni anatomiche e funzionali può perfettamente risentire e riprodurre le voci misteriose" of these humble creatures. This recalls Delbœuf's theory of love—that the sovereign wills of an ovule and a spermatozoön impel the youth and the maiden to seek each other's company. The lecture is not so serious a piece of work as that just noticed; the psychology in it is very incomplete.

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[1] "Di alcuni fondamenti fisiologici del pensiero," Milano-Torino, 1890. (*Rivista di Filosofia scientifica*, April.)

[2] Trieste, 1893. ("Raccolta di scritti di medicina per côlti profani.")

Similarly defective in general psychological grasp is Professor Brugia's "Pensiero e senso organico."[1] This is the greater pity as the writer's use of method in weighing evidence seems more than averagely good. He begins by emphasising the solidarity of the organism: though the viscera are controlled by special centres, and act and react on one another with a certain degree of independence, yet in the last resort the highest-level centres are the appeal, and they have the final responsibility. Mind is "una forza insita nel cervello," which is at the same time "l'organo della recettività cenestesica." Or again, mind is "la sintesi di tutti i fenomeni somatici." To the question of the nervous connections between viscera and brain the author has no answer of his own; he carefully reviews the evidence, and concludes that we have certain knowledge only of the points of arrival and departure. He emphasises the psychological value of cœnæsthesia: "è sopratutto l'oscura coscienza organica che dà colorito alle impressioni e alle idee, che . . . determina il modo d'essere e di reagire delle cellule nervose corticali." The psycho-organic co-ordination becomes less complete as mentality develops. The doctrine of temperaments is set forth under the rubric "influence of the cerebral cortex on the organic constitution;" and then follows an important chapter on visceral disturbances in the ætiology of psychoses. Dr. Brugia holds what may be called a waste product theory ("una possibile origine tossicoemica," "un ricorrente processo di autointossicazione della corteccia," are characteristic phrases). It is curious to find this hypothesis based upon the analogy from sleep. Surely a waste-product theory of sleep, whether the product favoured be lactic acid or carbonic acid, cannot avoid the necessity of a supplementary hypothesis, introducing the play of an inhibitory mechanism. And a bolstered theory is never quite satisfactory. As a set-off to this slip I cite a remark which is too good to be overlooked: "Per ciò che concerne là delimitazione delle influenze somatiche che agirebbero 'per contraccolpo' sul cervello," writes the author, "è chiaro che . . . un profondo studio di molte psicosi che hanno apparamento forma reflessa potrà scoprire qualche nuovo fattore genetico, insieme à una condizione che, per essere la piu conspicua, può sembrare la sola operativa." The only other general chapter deals with the organic evolution of instinct. The views of Darwin, Spencer and Romanes are discussed with some acuteness; but there is no indication that the writer has put the question as to what an instinct *is* psychologically, *i.e.*, what processes a cross section of the instinctive consciousness would show. Yet the psychology of instinct is at least as important to his general enquiry as the biology of it. The rest of the book is mainly taken up with casuistry. There are chapters on psycho-organic disturbances of sex, on similar disturbances of the instinct of nutrition, on the signification of the so-called "internal hallucinations," on cœnæsthesia and impulse, on cœnæsthesia and delirium, and on the motor power of the organic senses. A general summary of their results would have been serviceable.

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[1] "Saggio di psico-patologia della cenestesi," Aversa, 1893.

It is somewhat late in the day to offer a review of the last work to be noticed—M. Paulhan's "L'activité mentale et les éléments de l'esprit."[1] The book is essentially an attempt to better the associationist psychology, made by an associationist psychologist. The new concept introduced is that of the "system"; we have a law of systematic association and a law of systematic inhibition. The former runs: every psychical fact tends to associate with and to call up psychical facts which are harmonisable with itself; which can co-operate with it for a common end or for harmonic ends; which can form with it a system. The latter: every psychical phenomenon tends to prevent the production, to arrest the development, or to cause the disappearance of psychical phenomena which cannot unite with itself in accordance with the law of systematic association, that is to say, for a common end.

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[1] Paris, 1889.

Reviews and Abstracts.

Traité pratique des Maladies du Système nerveux. Par J. Grasset et G. Ranzier, Professeurs à l'Université de Montpellier. Montpellier: Camille Coulet. Paris: G. Masson. 1894. Price 90 fr. Fourth edition, with 122 figs. and 33 plates. Two vols., 8vo, 900 and 1,090 pages.

It is exceedingly difficult to appreciate at its proper value such a monumental work as the treatise of Grasset and Ranzier in its most recent form. The only plan of arriving at even an approximate estimate of its value is to take here and there a section, and regarding such a section as a separate work on the subject with which it deals, to see to what extent it meets the demands which one is accustomed to make on a monograph. For many of the sections viewed in this manner no praise is too high, while others are a little disappointing in their sketchiness.

The plan followed in such a work is of no little importance, and it may be useful if we first indicate what this is, for in this way its defects and its advantages will be obvious. There are in all six parts, the first dealing with brain disease and including circulatory troubles and local diagnosis, encephalitis, tumours and hydrocephalus. The second part deals with spinal cord diseases —myelitis, diffuse myelitis, diffuse cerebro-spinal disease and circulatory troubles; the third part discusses bulbar diseases; the fourth, diseases of the meninges—cerebral and spinal; the fifth treats of nerve diseases and neuroses; and the sixth of nervous manifestations of general diseases. No doubt such an arrangement has much to recommend it. It is probably as comprehensive as any, and to say that it is unscientific in its arrangement is to apply to it a criticism which is equally applicable to every systematic work on diseases of the nervous system that has yet been produced. And the retort generally made to such a criticism is no doubt true, viz., that the state of our knowledge of nervous disease, especially in its pathological aspects, is not yet such as to permit of such a scientific work being produced. The arrangement used possibly leads occasionally to the separation of intimates, as when Jacksonian epilepsy and so-called idiopathic epilepsy are found in different volumes, and, like misfortune, it occasionally makes strange bed fellows, as when tetanus and paralysis agitans are found on neighbouring pages. But in spite of such drawbacks the arrangement is a clear and convenient one, and, as we have said, it is certainly comprehensive.

We have thought it best in order to indicate the general character of the book to take one chapter, and by a brief *resumé* of its contents to show how complete and how well arranged it is. Taking as a good example the chapter on syringomyelia, we find first of all a short but succinct history of this condition from the first use of the word, in 1837, down to the time of publication of the work under notice, and in this is pointed out the curious anomaly that it was known as a pathological condition long before symptoms were recognised as associated with it, and that the clinical recognition of the disease has led to a reconsideration of at least some points in the physiology of the spinal cord. The pathological anatomy of the condition is next dealt with, the appearance of the cord, the number of cavities, their contents and their walls are described and illustrated with a series of beautiful plates, and the relation of the cavities to the central canal is fully discussed, as well as the manner in which they arise. The condition as a clinical one is next considered, and a brief history of its recent discovery is given, and its symptoms both intrinsic, *i.e.*, peculiar to this condition, and extrinsic, those, so to speak, accidentally associated with it, are fully set out. The former comprise the peculiar sensory dissociation, the muscular atrophy and the trophic changes; the latter such conditions as frequently exist in other diseased conditions such as lateral sclerosis and posterior sclerosis. Differential diagnosis is next considered: (1) From such diseases as usually show no sensory impairment, *e.g.*, progressive muscular atrophy, myopathies, amyotrophic lateral sclerosis, cervical pachymeningitis, disseminated sclerosis, tabes, chronic myelitis and compression of the cord by tumour or bone disease; and (2) from diseases in which sensory symptoms are predominant, *e.g.*, neuritis, leprosy and hysteria. In this part is introduced a subsection on the maladie de Morvan, in which are set out the different views held as to the relation of this disease to syringomyelia and leprosy, and while no definite side is taken, it is, we venture to think, indicated that the authors regard the maladie de Morvan as merely a form of syringomyelia. The article is concluded with a short account of the etiology, the prognosis and treatment. *Ex uno disce omnes*, and if all are not equally good not one can be said to be bad. Further criticism than this is unnecessary except that we may say that the style is characterized by that dramatic clearness and force which brings the type of disease before the reader in all its vivid reality—a characteristic which is so happily evident in the great bulk of French medical writing. The illustrations are good and copious, in no case unnecessary, and as a rule happily chosen to illustrate and complete the text, and the work as a whole is one in which the authors may have a just pride, and in which the reader will have unlimited enjoyment.

JAMES TAYLOR.

Les Altérations de la Personnalité. Par ALFRED BINET, Bibliothèque Scientifique Internationale. Paris: Félix Alcan, 1892.

THE author points out that of late years a large body of observers have been working at pathological psychology through hysteria and hypnotic suggestion. As researches have multiplied numerous discussions have taken place, not only as to the interpretation of facts, but as to the facts themselves. The author's object is not to bring forward his own observations, but to select out of the crowd of researches those which have received confirmation at all hands, can be gathered into one synthesis, and which lead to one conclusion. The time is ripe for such an attempt, for it is a curious fact that a number of observers in various countries, working by diverse means with different ends in view, have consciously or unconsciously arrived at the same conclusion, namely, that underlying the mental phenomena of hysteria and suggestion is a peculiar alteration of consciousness, a doubling or rather a parcelling out of the ego.

In working out his thesis M. Binet takes first, under the divisions of spontaneous and induced somnambulism, cases of dual consciousness, alternating in time.

Spontaneous somnambulism is especially important as its phenomena are not influenced by preconceived notions on the part of the observer. In this condition the subject leads another psychological existence or second life, of which he has no memory on returning to the normal state, and in which he lives the common life, being open to all ideas and perceptions, and has no delusions. Two mental lives are thus lived side by side; in the first state there is no memory of the second and *vice versa*. Several celebrated cases are referred to. In Félida X., there was loss of memory of the second state when she was in her normal condition, but in the second state she had memory of both the normal and abnormal lives. A change of character also characterises this second condition, which may further be attended by sensory or motor defects. An observation of M. Proust makes known the important fact that the memory during induced somnambulism extends to events of the second state (spontaneous somnambulism). So far these cases have been observed in hysterical patients only, but if changes in memory, which are always more easy to appreciate than those of character, be taken as the touchstone, the author thinks similar phenomena may be observed outside hysteria.

In the next group of cases the second state is altogether different from the normal one; the subject is under the influence of one idea or group of ideas, and is unconscious of his surroundings unless they fit in more or less with the preconceived idea which dominates his thoughts and actions. Mental activity is thus developed in one direction only. This activity has been considered to be unconscious, purely reflex or *machinal*, without trace of conscious thought, judgment or imagination. The author does not accept this view and brings forward data to show the presence of consciousness. Subjects in this second stage may not be able to speak or comprehend speech, but in a patient observed by Charcot this was not the case, and by means of speech the nature of the patient's delusion could be ascertained. The difference from induced somnambulism consisted in the spontaneity of the delusions which could not be guided at the will of the operator. In this class of cases there is dual consciousness, but the second state is attended with delusions. It is, therefore, more difficult in cases of this class to be certain whether, as in those of the first class, memory during the second state extends to all events of preceding phases of this state, but the occurrence of continuity of action during successive phases seems to prove that it does.

Induced somnambulism or the hypnotic state constitutes from the psychological stand-point a new existence—it is the emergence of a new personality. In this form of somnambulism alterations of character are not observed, but the memory shows profound change; first, the subject during the normal life can recall none of the events of the hypnotic state, and secondly, whilst in the latter he remembers not only the events of former periods of somnambulism but also those of his normal life. Memory in the induced somnambulistic state is thus more extensive and also more exact as regards detail than in the normal life. In this respect spontaneous and induced somnambulism correspond.

The advantage of induced somnambulism is that it enables the experimenter to vary the conditions, and study somnambulistic phenomena more completely. It has thus been shown that the normal life and the somnambulistic state alternate with each other, and that during the former there is no memory of the events of the latter. Gurney has shown that though the subject in the normal is ignorant of the events of the somnambulistic state, and cannot recall them by any effort of the will, yet by putting a pencil in his hand, which is concealed from him by a screen, the hand will automatically write words heard during somnambulism, and further will correctly finish an addition sum begun in the somnambulistic state. The hand writes; the subject is unaware of what is written. All the cases hitherto considered have this in common, that a set of psychological phenomena well co-ordinated amongst themselves form a group outside and apart from the normal consciousness. In natural somnambulism the new or secondary consciousness alternates with or replaces the normal consciousness; in the last experiment it co-exists with it.

Part ii. deals with double consciousness, and is entitled "Co-existent Personalities." Double consciousness is met with under two conditions: (1) in hysterical anæsthesia; (2) in strong concentration of attention on one object. Activities may take place without the ordinary consciousness being aware of them, in the first in the anæsthetic regions and in their correlated nervous centres, and in the second in those parts of the body and their related nerve centres away from which the attention is directed. These secondary activities may show psychical characteristics (intelligence) and so come to form a secondary consciousness existing by the side of the normal one. It is true that automatic actions occur frequently in ordinary life, but they are simply habitual actions, arise from frequent repetition of movements, rarely attain any marked degree of development, and are not therefore well adapted for the present study. Hysterical anæsthesia is to be regarded as a psychical anæsthesia arising from mental dissociation. This can be shown by experiment. In all dealings with hysterical patients due precautions must be observed in order to avoid making suggestions. Hypnotism is unnecessary. All that is required is to place the anæsthetic limb, *e.g.*, the hand, behind a screen or behind the patient's back and to be careful not to speak to her. Then the hand will carry on single movements imparted to it, will assume the writing position if a pen be placed in it, and further, if it be passively made to carry out a writing movement, on letting it go the word or character may be written. If the hand has been caused to trace a word wrongly spelt, it may, strange to say, when it reproduces the word, be seen to hesitate at the wrong letter and either reproduce the mistake or correct it. Of all these movements, passive and active, the subject is entirely unaware.

In some patients more complicated actions can be evoked than in others. Women, especially those who have been frequently hypnotised, are better subjects than men; probably this is due to the difficulty of eliminating some form of suggestion. The above facts indicate that in hysterical anæsthesia a real perception is gained by the patient, which does not, however, rise into the ordinary consciousness, and since phenomena of memory and judgment have been shown to occur, the existence is presumed of an intelligence different from the ordinary one and acting without knowledge or aid of the latter.

In another form of experiment if the hysterical subject be placed before some test types, but just beyond his distance of distinct vision, although he cannot read them, his hand may automatically write them correctly. The author attributes this to feebleness of the sensory (receptive) organs, and remarks that the "threshold of consciousness" is in hysteria a relative term, excitations below a certain minimum of consciousness being registered unconsciously, and resulting in subconscious actions.

The effect of attention is to exalt the sensibility of the senses engaged in the special work, to lower that of all others coincidently. The distraction resulting from attention is a temporary anæsthesia; hysterical anæsthesia is a permanent distraction. In normal persons, however close the attention, actual anæsthesia is never produced. In the hysterical it is readily produced, and according to Janet this results from "the restriction of the field of consciousness." The secondary consciousness or personality produced in the above ways: (1) perceives sensations over anæsthetic areas; (2) remembers phenomena of similar former states; (3) seems to have a distinct personality; (4) responds extremely readily to suggestion. The identity of this secondary consciousness with that of somnambulism is shown by the state of the memory. So far the most important conclusion reached is the division or dissociation of consciousness, *i.e.*, the juxtaposition of several psychological states which are never confounded together.

Hysterical patients can often use an anæsthetic limb, and contrary to what one would expect can write when their eyes are closed, and their right hand is completely anæsthetic. As under these circumstances they have no knowledge of what is passing, they judge of how much has been written mainly by lapse of time; thus, if asked to write one character a dozen times in succession they rarely succeed in writing it the correct number of times. The author explains these actions by (1) the preservation of a good visual memory, and (2) the survival of the afferent impulses and motor images in a separate consciousness; he does not think that they can be explained by the theory of a sense of effort conditioned by the outgoing current. Some hysterical persons are unable to carry out any action without the aid of sight, attributed partly to feebleness of visual memory and poor development of secondary consciousness, partly to the fact that light appears to be a necessary physiological stimulus to the enfeebled nervous system. Though the primary and secondary consciousness are separated, yet experiments show that there are important connections between them, that they inter-act one on the other; for instance, an hysterical person asked to recall a name or date may be unable to do so, although it is on the tip of his tongue; in this case it may at once be written without his knowledge by his anæsthetic hand, thus showing collaboration of the primary with the secondary consciousness, and that memory is more extensive in the latter. There is thus no abrupt demarcation, or suspension of all relations between the primary and secondary personalities; each group of psychological ideas influences each neighbouring group, but is only itself cognizant of what is passing in its own domain. Although it has been so far assumed in order to make the argument simpler that the subject is entirely unconscious of what passes in the anæsthetic limb, this ideal is rarely attained. Any excitation of an anæsthetic limb can arouse by means of suggestion conscious ideas; M. Binet thinks that this is accomplished through the intermediary of the secondary consciousness which then influences the primary one. Sensations produced in the anæsthetic limb remain subconscious, but he shows by experiments that they may give rise to ideas, images, false perceptions or hallucinations in the normal consciousness, of whose real origin the subject remains entirely ignorant.

The third part of the book deals with alterations of consciousness (of personality) produced by suggestion.

Fictitious personalities may be created by suggestion. Under the influence of the suggested personality the subject ceases to perceive the external world, and falls under an hallucination. Everything in harmony with the suggestion is evoked, and everything that contradicts it is inhibited, and passes out of consciousness, hence there is amnesia as regards the former life, so that the memories which represent the past existence and form the basis of the ego are temporarily effaced. A division of consciousness is thus made, and a new personality is formed. Two classes of patients exist: in the first their real personality is not lost sight of, but they cannot resist the ascendancy of the operator, the others are completely victims of the illusion suggested. Again, former stages in the life of the subject may be recalled, he may be thrown back into his condition at a past period of his life, and reproduce his behaviour at that age. From the psychological point of view these experiments show that ancient memories which we believe lost because we cannot recall them at will, live on in us; in other words, what we know of ourselves is only a part of what we are. This recall of phases of the past life by suggestion cannot be explained by the law of association of ideas, for if it could be there would be no need of suggestion to provoke the recall. The author argues that if, then, the law of association is not sufficient for this recall, it fails to fully explain the development of the mental life. Causes more profound of whose nature we are hardly aware act to gather up our ideas, perceptions and memories into autonomous and independent syntheses.

Passing on to the discussion of the interesting subject of systematised anæsthesia by suggestion, by which term is meant the condition which results when a suggestion is made to the subject that he shall not perceive a certain object, the author compares this condition to that of spontaneous hysterical anæsthesia, the perception inhibited by suggestion having the same fate as sensations coming from an anæsthetic area, and like them being relegated to a secondary consciousness. Certain facts show that the object inhibited by suggestion is really seen. (1) A square of red paper not seen (obediently to suggestion) calls up an after sensation of the complementary colour. (2) The subject is told not to see one out of a number of similar cards; if they are too much alike mistakes are frequently made, but no mistake occurs if "*invisible*" be written across the card which is not to be seen. Perception and judgment must therefore be present, but are unconsciously exercised, and systematised anæsthesia is preceded by a certain number of subconscious psychological processes.

All negative suggestions do not, however, act by means of a dual personality in this way, although they do so in persons who have been repeatedly hypnotised; in those hypnotised for the first time nothing of the sort occurs—a certain perception is excluded from the sphere of consciousness and that is all—and it must be conceded that much obscurity still surrounds the *modus operandi* of negative suggestion. In discussing spiritualism the author for obvious reasons feels obliged to restrict himself to the facts which consist in the unconscious movements executed by the so-called medium. He brings forward evidence of the existence of dual consciousness in mediums. The peculiar phenomena of spiritualistic manifestations which cause them to differ from those of double consciousness previously studied are due to the fact that the medium is not ignorant of the end in view, like the foregoing hysterical subjects, but is dominated by a powerful preoccupation from his belief in spirits and in the possibility of evoking them. He is thus placed under the most favourable conditions for auto-suggestion. Curiously, however, it is the secondary and not the primary (normal) consciousness which is influenced by the suggestion. Whereas in previous parts of the book division of consciousness is due to hysterical anæsthesia or to distraction, in this case it can be traced to auto-suggestion. The mental modifications are here more complex and less easy to describe than in the first two.

M. Binet concludes: (1) That the elements which normally constititte the ego may be in a state of division or disaggregation.

(2) A consciousness does not cease to accompany these elements, although the primary ego loses knowledge of them.

(3) Sometimes under exceptional conditions, pathological or experimental, these elements are organised into secondary personalities.

The foregoing will give some idea of the main argument and scope of the work. M. Binet, it will be seen, traces the condition of dual or multiple consciousness from the form in which it is most easily observed—alternating states of double consciousness—to those more complex forms in which two or more distinct personalities exist side by side. He shows that this condition may occur spontaneously or be artificially induced. Dual consciousness seems to exist in a more complete and highly developed form when it occurs spontaneously. So far as can be judged due care has been exercised not to admit any observations into the book which are not well authenticated. The whole of the investigation into dual consciousness during recent years, the main facts of which are given in this book, is exceedingly important from the new light thrown upon the nature of consciousness. Under the conditions mentioned, hysteria and somnambulism, there seems to be a firm basis of fact for the splitting up of consciousness into subordinate parts. To what extent may this subdivision of the mind occur under normal conditions? So far it has been shown to appear in only a rudimentary and imperfect form during concentrated attention in normal persons. One of the most important features of these researches lies in their bearing upon the nature of hysterical anæsthesia, as has been mentioned above.

It only remains to be said that the book gives a most clear and concise account of a difficult subject. The author has marshalled his facts in orderly sequence so that the reader can readily follow and appreciate the line of argument, and the work is one which will amply repay a careful perusal.

J. MICHELL CLARKE.

Recherches Cliniques et Thèrapeutiques sur L'Épilepsie, L'Hystérie, L'Idiotie et L'Hydrocéphalie. Par BOURNEVILLE, Médecin de Bicêtre, avec la collaboration de MM. BONCOURT, CORNET, LENOIR, JULES NOIR, et P. SOLLIER. Vol. xiv., avec 89 figures dans le texte et une planche. Paris, 1894.

THE book is divided into two parts. The first gives an account of the work that has been done at the Bicêtre and Fondation Vallée during the year 1893; the second consists of eight essays by Dr. Bourneville and his assistants on subjects of special interest.

Dealing with the first part we find that various improvements have been made in the mode of teaching the children, especially with reference to the education of the eye, the hand, and the faculty of attention. During the year 122 cases were received, 15 died, 33 were discharged, 4 were transferred to other parts of the Bicêtre, and 5 escaped, leaving on December 31, 1893, 512 patients. The causes of death are related, and a table giving the weights of the thymus and thyroid glands is of great interest. The Fondation Vallée is overcrowded, and contains no less than 131 children, but a project is on foot to increase the size of it by building a pavalion capable of accommodating 100 more patients. In this institution, too, various improvements in the mode of teaching have been made, and exercises for improving the power of the hands and legs have been added.

One of the most important and at the same time interesting essays in the clinical part is that by Dr. Bourneville himself, who contrasts the surgical with the educational treatment of idiots and imbeciles. He analyses eighty-two cases in which craniectomy has been practised, and comes to the conclusion that the results have been slight or doubtful. The operation has been performed by Dr. Lannelongue with the idea of giving free scope to the brain by weakening the resistance of the cranium. He supposes that microcephaly, a generic term under which he describes the different forms of idiocy, is due to premature ossification of the cranial sutures. Dr. Bourneville goes carefully through a series of twenty-two cases, in three of which craniectomy had been practised, and finds no sign of premature synostosis, and he concludes that Dr. Lannelongue's theory is not borne out by pathological anatomy. Moreover, lesions of the brain, which are often found, cannot benefit by the operation. Dr. Bourneville then relates eleven cases in which educational treatment alone has been carried out, and shows very clearly that more good will be effected by this treatment than by craniectomy.

An essay by Bourneville and Cornet on thirty cases of epilepsy, treated by subcutaneous injections of testicular fluid, is also very instructive. Excluding two cases who did not undergo the full treatment, there remain twenty-eight who were injected with the fluid for a period of six weeks to two months. According to M. d'Arsonval, if at the end of six weeks there are no results in the treatment of epilepsy, it is useless to prolong it. Of the twenty-eight patients eight had a slight diminution in the number of their fits, while the remaining twenty had a larger number than before. In no case had there been amelioration of the mental condition. The weight was taken before and after the treatment in each case; unfortunately in several cases the weights have been mislaid, but of ten patients in which these were preserved there was found to be an increase in six, a diminution in three, and no change in one case.

A long essay of nearly two hundred pages on some forms of hydrocephaly, written by Drs. Bourneville and Noir, is interesting. The authors distinguish three forms, viz., simple hydrocephaly, scapho-hydrocephaly, and symptomatic hydrocephaly, and give the histories of nineteen cases, eleven of which illustrate the first, five the second, and three the third form of the disease. The symptoms generally found are fully described, and the condition mentally, as to language, will, the instincts, and moral and social sentiments, is well delineated. According to the authors, in most cases the effusion of fluid takes place some months after birth, and the child, to all appearance in a normal condition of health, is suddenly taken with meningitis or convulsions. Gradually the head increases in size until the age of 4 or 5 years, when its greatest dimension is reached. Death usually takes place between the age of 3 and 6 years from broncho-pneumonia or meningitis. As regards the etiology, all cases of chronic hydrocephalus have as predisposing causes nervous diseases, mental maladies, and alcoholism; the exciting causes being chiefly chronic inflammation of the intra-ventricular meninges or compression of the vessels of the base of the brain by the development of a tumour. The authors do not believe that treatment by trepanning and drainage is of any use, as the result in most cases is fatal; treatment by compression of the head, daily friction with mercurial ointment, and the administration of calomel, aided by the use of salt baths, massage of the limbs, and tonic medicines, being far more successful.

The book is profusely illustrated with wood-cuts of many skulls and brains, and of a few patients who have been under treatment, and shows that Dr. Bourneville and his assistants continue to do much excellent work in their department of the Bicêtre.

FLETCHER BEACH.

Psychiatrie für Aerzte und Studierende. Prof. TH. ZIEHEN. Berlin: F. Wreden, 1894. Pp. ix. 470.

In this work, which forms one of a series of medical text-books, the author treats diseases of the mind on the basis of the psychological doctrines laid down in his "Leitfaden der physiologischen Psychologie," of which an English translation has appeared. One of the marked features of that book was the thorough-going adherence to what the author called the "association psychology of the English," though his system was distinguished from that of English psychologists by the importation of the Herbartian notion of the mutual strengthening or inhibition of ideas to which the name of "constellation" was given. The first part of the present book, entitled general psychology, extends over 150 pages, and is devoted to an account of the author's psychology with its application to the analysis of abnormal mental symptoms, and it is this part of the book which will be chiefly interesting to neurologists. In it mental symptoms are considered under five heads as disturbances of sensation, of memory images, of intellectual feeling tone, of association of ideas, and of action. Under the first heading illusions and hallucinations are chiefly considered, the latter term being limited to those cases in which there is no actual stimulus external to the body. A novel feature is the division of both illusions and hallucinations into two groups: those in correspondence with the general content of consciousness, and those which have no relation to this content, but arise out of latent or half-forgotten memory images. It is doubtful whether this division, though corresponding to a psychological distinction, is one of practical value.

The section on affections of memory images is chiefly devoted to word blindness and deafness, and these conditions support the author in the opinion expressed in his former book, that the memory images have a physical basis different from that of the original sensations—a view which is gaining ground among psychologists.

In the section on affections of intellectual feeling tone, exaltation, depression, and other emotional conditions are considered. Much importance is attached to irradiation of feeling tone, whereby one element of a mental complex with a pronounced painful or pleasurable tone of feeling transfers this tone to the whole.

The next section, on disturbances of association, is an important one. The relation of increased or lessened rapidity of the flow of ideas to the state of feeling is considered at some length, being of importance in its bearing on the classification adopted later by the author. The subject of incoherence or dissociation of ideas is treated in a specially interesting manner; incoherence is regarded as frequently secondary to other psychopathic conditions, such as increased rapidity of flow of ideas, hallucinations or delusions, violent emotions or weak-mindedness; but the author insists on the existence of a primary dissociation, depending on a disturbance of association paths. Such cases of incoherence are analogous to those cases of aphasia caused by lesions of paths connecting different centres, which may indeed be regarded as special forms of primary dissociation. This primary dissociation may affect not only the train of ideas, but may occur between the sensation and idea causing defective orientation in regard to surroundings, or it may occur between the ideational and motor elements, giving rise to incoherence of action.

In the section on morbid actions, the author refuses to regard such actions as dependent on disturbance of any special will process, and sees in them only the natural outcome of the disturbed sensory or ideational processes.

In the chapters on diagnosis, ætiology and therapeutics, the author shows his close acquaintance with the more practical side of his subject. The chapter on diagnosis has an excellent outline of symptoms as a guide to the examination of patients.

The chief interest, however, of the latter half of the book will be found in the classification adopted. This differs considerably from those most usually met with in England, and chiefly in the treatment of the simple or primary forms of insanity. Professor Ziehen takes the psychological distinction between cognition and feeling as the basis of his classification. The term "mania" is limited to cases in which the general tone of feeling is altered in the direction of pleasure, a condition the true opposite of melancholia when the alteration is in a painful direction. In both mania and melancholia the change of feeling is primary and the intellectual disturbance present is secondary to the state of feeling.

Cases, on the other hand, in which the primary and dominating disturbance is of the cognitive side of mental life, form a group to which the name paranoia is given, and this includes many, if not the majority of the cases which in England would be labelled "mania." This has four chief sub-groups: acute and chronic simple paranoia, acute and chronic hallucinatory paranoia. In the latter forms illusions and hallucinations are the chief symptoms, delusions developing from these, especially in the chronic variety. In the simple forms delusions predominate, chronic simple paranoia corresponding to the chronic delusional insanity of English writers. These four chief forms have their varieties: acute hallucinatory paranoia, for instance, having varieties depending respectively on the predominance of increased or lessened rapidity of mental processes, of incoherence, of exaltation, or of depression.

The book concludes with an excellent series of plates illustrating the various types of insanity. Professor Ziehen may be congratulated on this addition to the text-books of insanity. It is valuable especially for the general coherence which has been given to one of the most confused branches of medical knowledge, and this coherence is mainly due to its foundation on the basis of a systematic psychology. Whether this psychology is a true psychology is another matter. Whatever views may be held about it it must be acknowledged that it may form a useful basis for the study of mental pathology.

W. H. R. RIVERS.

Hérédo-Ataxie cérébelleuse. Par le DOCTEUR PAUL LONDE. Paris, 1895. Battaille et Cie. By J. A. Ormerod, M.D.

THIS is an interesting monograph on a disease which, as yet, is little known. The author begins his work by remarks on "family" diseases, and more particularly family diseases of the nervous system. By a family disease we are to understand one which tends to attack many members of the same stock, and particularly, it would appear, members belonging to the same generation, and which depends not so much on extrinsic causes, whether these be intra-uterine or subsequent to birth, as upon a fault inherent in the germ or introduced into it by procreation. Attempts to classify such diseases according to the rough anatomical divisions of the nervous system (cerebrum, medulla, cord, nerves, &c.) do not give very satisfactory results; hence the author sketches out a classification based on nervous functions and on the "systems" of fibres and centres connected with such functions. Thus the cerebral motor system (highest motor neurons) is affected in the diplegia of Freud and Little, and in the spastic paraplegia of Strümpell; the spinal and peripheral motor system in certain forms of muscular atrophy; the cerebello-spinal system, which is devoted to the function of equilibration, in Friedreich's disease, and in hereditary cerebellar ataxy.

This name (*hérédo-ataxie cérébelleuse*) proposed by Marie, who collected and critically studied cases of the disease, is intended to indicate three main facts:—(1) That it is a family disease; (2) That ataxy, or at least the staggering gait known as cerebellar ataxy, is a leading symptom; (3) That the lesion is in the cerebellum. By this last factor it is distinguished from

Friedreich's disease, where there is also a family ataxia, but where the lesion is spinal. Yet the two diseases are closely related; both consist in a degeneration of the cerebello-spinal system, beginning in Friedreich's disease at the spinal end, in the present disease at the cerebellar. This is illustrated in Menzel's case, wherein both spinal cord and cerebellum were degenerated; the two diseases here co-existed, or rather were exhibited in their proper pathological unity.

The description of the disease is based on the following cases, of which the author gives details in an appendix.

A. Cases collected by Marie; viz., 2 recorded by Fraser (*Glasgow Medical Journal,* 1880), 3 by Nonne (*Arch. für Psychiatrie,* 1891), 8 or more by Sanger Brown (BRAIN, vol. xv., 1892), 3 by Klippel and Durante (*Revue de Médicine*).

B. Original cases; 1 by Brissaud and the author (*Revue Neurologique,* 1894), 2 by the author (*Revue Neurologique,* 1894).

C. Doubtful cases; 2 by Erb (*Neurologisches Centralblatt,* 1890), 2 by Seeligmüller (*Archiv für Psychiatrie,* vol. x.).

There is also the case (just mentioned) by Menzel (*Archiv für Psychiatrie,* 1891); and some extremely doubtful cases by Hervouet (*Gazette Médicale de Toulouse,* 1891) and by Botkin (*Revue Médicale de Moscow,* 1883).

The leading fact in the etiology is the family predisposition. How this predisposition is brought about we know not. Tubercle appears to be not infrequent in such families, and, as in Friedreich's disease, parental alcoholism has been noticed, though whether this is really a cause remains doubtful. Parental syphilis and parental consanguinity do not appear as causes. Little is known as to exciting causes; neither traumatism nor infectious diseases have, in the author's opinion, much influence; in one case (Nonne) the disease followed exposure from shipwreck. The age at onset is comparatively late, usually after twenty; in one case it was past forty. This may explain why the disease appears in successive generations more frequently than does Friedreich's disease, where the earlier onset is more likely to prevent marriage. As to the relative frequency of occurrence in male and female, and of transmission through male or female, we can as yet hardly be certain.

Stated generally, the symptoms are as follows:—An unsteadiness in walking and standing appears gradually and slowly progresses, being sometimes accompanied by pains in the legs and loins; after a variable time (usually one to three years), the movements of the hands become uncertain, and the speech and the eyes become affected in a manner that will presently be described. The tendon reflexes are preserved or exaggerated; sometimes there is actually contracture of the limbs. The mind sometimes deteriorates. The disease is progressive, though there may be remissions; death is mainly due to intercurrent disorders.

The author proceeds to a more minute description.

Motor Symptoms.—The gait is cerebellar, *i.e.*, the patient walks with feet apart, swerving and swaying from side to side, as if intoxicated. This is mainly due to difficulty in maintaining the equilibrium of the trunk, but there may also be, particularly in advanced cases, a true inco-ordination of movement in the legs. In standing there may also be unsteadiness, but this is not increased by closing the eyes (no "Romberg's symptom"). The earliest defect in movements of the hands is a wavering or hesitation as the hand nears its mark; then a tremor or oscillation which shows itself towards the end of a voluntary movement. Fine actions, particularly that of writing, are the first to suffer distortion; later a tremor affects coarse movements, such as that of lifting a cup. Apart from this tremor of the hands, which may be compared to that of disseminated sclerosis, the patient exhibits involuntary jerky movements, like those of chorea, in his limbs, trunk, head or face. Another peculiar motor symptom is a prolongation of muscular contraction, so that the patient cannot, when he wishes, let go an object he has once grasped. The face exhibits what may be called an exaggeration of associated movement. Thus, during any natural facial movement, but particularly while speaking, the occipital frontalis is thrown into violent action, and the eyes open widely, so that the patient has a staring, astonished look; the naso-labial fold becomes exaggerated, the mouth may even be drawn into a tetanic grin. The speech is remarkable—not syllabic, nor hesitating and elisive; but jerky and explosive, words and syllables being brought out with sudden and unnecessary vehemence. The voice is usually low, guttural and monotonous; or it may break suddenly into high notes. The head is unsteady, particularly when the patient is excited; or there may be a definite spasm of the neck-muscles, causing torticollis. In some cases there has been marked vertigo. The tendon reactions are normal or exaggerated; the muscular sense is normal, the muscular strength is normal.

Sensory disturbances, if present at all, are vague and subjective, *e.g.*, headache, pain in the loins, numbness in the legs, in one case shooting pains.

Trophic disturbances are practically absent; it seems doubtful whether spinal curvature and club-foot may not sometimes occur. The expression (except for the over-action of the facial muscles just mentioned) is dull and dejected; memory and intelligence are sometimes dulled, the disposition may be irritable.

Remarkable *ocular symptoms* may occur; thus, as regards the fundus oculi, optic atrophy of very various degrees has been observed, and its concomitants, amblyopia, and contraction of the field of vision. As regards ocular movements—during fixation there is a sort of instability of the eyeballs, a condition of irregular, jerky movement, resembling the choreiform movements of the limbs rather than true nystagmus. In several instances there has been a paresis of the R. external rectus, sometimes of the R. superior rectus. Sanger Brown noted a peculiar droop of the upper eyelid, existing only when the face was at rest, and giving place during animation of the face to overaction of the levators and occipito-frontalis, which causes a peculiar stare. There are no particular *visceral troubles;* menstruation comes on late, probably from a general backwardness of development; certain difficulties of deglutition and of micturition recorded by Sanger Brown are explicable (our author thinks) by an ataxia of the muscular movements concerned, in fact, ataxia and instability constitute the keynote of the symptomatology.

A few remarks, which must be regarded as provisional, are made as to the possible stages and varieties of the disease. There may be prodromata of a vague character, "neurasthenic" they may be called; the first stage (ataxia) is marked by the reeling gait; the second (astasia) by such increase of inco-ordination as prevents walking or standing alone, and by the appearance or aggravation of the disease in the hands; the third (impotence) by general helplessness and disability from aggravated ataxia, rigidity, optic atrophy, &c. The author thinks that two principal forms may exist, consisting of ataxia with, and ataxia without, ocular symptoms, and that there may be also characteristics special to each family, such as the peculiar ptosis noticed by Sanger Brown, the facial asymmetry, mental deterioration, &c., by Nonne, vertigo by Fraser, hysteria by himself.

About the pathology much remains to be learnt. Our author holds, with Marie, that the essential anatomical fact is atrophy of the cerebellum. But there are only a few *post-mortems*, those by Fraser, Nonne, Menzel. Nonne found a widespread atrophy of the nervous system; Menzel's case showed degeneration of the cord as well as of the cerebellum. Still (as is pointed out in a chapter on the physiological pathology) the rough resemblance between the main symptoms of this disease, and those produced by other lesions, experimental or morbid, of the cerebellum, serves in some sort to designate the cerebellum as its seat. So far as we yet know, the character of the lesion is a pure and simple atrophy or disappearance of nerve elements, and therein differs from the cerebellar atrophy which follows traumatism or infectious diseases, wherein is found evidence of old meningitis and a sclerosis of the nervous centres from overgrowth of interstitial connective tissue.

The differential diagnosis from other kinds of family nervous diseases is considered. Obviously the nearest relative is Friedreich's disease. To save space we may, perhaps, refer our readers to a previous number of BRAIN (vol. xv., 270), for the clinical resemblances and differences of the two diseases. Dr. Londe lays special stress on the condition of the knee-jerks—absent in Friedreich's disease, present or exaggerated in hereditary cerebellar atrophy. Whether the knee-jerk is *invariably* absent in Friedreich's disease pure and simple we are not prepared to affirm. But, however that may be, we welcome as most suggestive the theory he advances, that both diseases consist essentially in a family tendency to degeneration of the cerebrospinal system, which strikes in the one disease mainly at the cerebellum, in the other mainly at the cord, and may sometimes, as in Menzel's case, involve both organs together.

Herter and others on Intestinal Putrefaction in Certain Neuroses.

HERTER[1] refers to his previous researches on intestinal putrefaction in epileptics,[2] and says that further researches have shown that excessive putrefaction in the intestines is met with in a variety of morbid states.

To determine the amount of this putrefaction he now makes use of Baumann's discovery that the various aromatic substances resulting from this putrefaction are after absorption eliminated through the urine as ethereal sulphates, and his observations are based on the quantitative estimation of these products.

In forty cases of epilepsy, two-thirds of the cases of grand mal showed distinct evidence of excessive putrefaction; in a few cases the excess seemed to correspond with the number of seizures, and reduction of the sulphates by treatment seemed to diminish the number of attacks.

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[1] *New York Medical Journal*, January 27, 1894.

[2] *Ibid.*, September 3, 1892.

Five out of eight cases of melancholia showed an increase of sulphates, but the author questions whether the increase might not be due to partial starvation, which is often present in these cases, for it is well known that inanition increases the putrefactive products.

In two cases of typical mania the sulphates were normal.

In six out of eight cases of chronic Bright's disease there was a marked increase of ethereal sulphates, and a considerable excess in many cases of simple anæmia, though in many the amount was normal.

In intestinal indigestion there was often an excess of putrefaction, and in intestinal flatulence, perhaps related to chronic catarrhal inflammation in the duodenum, indigo - blue was frequently met with in the urine.

"Many of the symptoms were related to the disturbances of nutrition that resulted in excessive elimination of uric acid, such excess being frequently found in intestinal indigestion associated with an excess in the ethereal sulphates, or with indigo-blue, or existing by itself."

The author suggests that the symptoms depend, to a large extent, upon the action of substances formed in the intestine during the putrefaction of proteids coincidently with the formation of the aromatic substances.

Excess of ethereal sulphates in the urine is evidence of excessive putrefaction of proteids in the intestine except when there is suppuration without free drainage; or except when certain drugs, as creasote and salol, are being given.

The regular occurrence of more than a trace of indigo-blue in the urine is pathological; it does not vary in proportion to the ethereal sulphates, but is due to a special form of putrefactive decomposition.

Intestinal putrefaction has as its most common symptom intestinal flatulence, but is not related to epigastric pain or tenderness, emptiness, or nausea.

Constipation and starvation increase putrefactive products in the urine, and epilepsy, acute melancholia, and chronic Bright's disease, are usually associated with excessive intestinal putrefaction.

Drug treatment of this putrefaction is unsatisfactory; but a diet of raw beef and milk, and the exclusion of vegetable nitrogen did good, and such diet reduces the excess of uric acid observed in many cases of intestinal indigestion, and part of the relief might be due to this.

Treatment of the intestinal condition was useful in epilepsy, and begun early in children might prevent the establishment of the epileptic habit.

Among those who spoke in the debate which followed the reading of this paper, Dr. L. C. Gray had got good results with a combination of salol, bicarbonate of sodium and pancreatin, and used also calomel and subgallate of bismuth, and Rochelle salts, in small doses for a long time. He regretted that the author had not given the amount of uric acid he found in these cases, as in his own cases, where patients were put upon a nitrogenous diet, there was always an excess of uric acid, and dietetic treatment had not given good results in his hands.

Dr. C. L. Dana had also found an excess of uric acid in almost all cases, but his treatment had been unsatisfactory. He recommended naphthalin for neurasthenic headaches to the extent of 60 to 80 grs. in a day.

Dr. W. H. Thomson agreed as to the frequent presence of intestinal derangement in epileptics, and their improvement when it was corrected.

He had not found any diet-list of use, but he believed in salol, or bismuth with benzoate of sodium.

Dr. L. Emmett Holt had had experience of intestinal indigestion in children, and for this he cut off carbohydrates and put them on a milk and meat diet, with an occasional dose of calomel when improvement was rapid.

MACPHERSON,[1] writing on disinfection in some forms of acute insanity, notices the very powerful effects of purgatives in aborting or cutting short attacks of recurrent insanity, and the very frequent association of gastro-intestinal troubles with melancholia, and argues that certain poisonous ptomaines resulting from putrefactive changes in the intestines might get into the circulation and so affect the nervous system.

He accordingly, in such cases, began by washing out the stomach and clearing the bowels with calomel with or without other purgatives, and on the second day of treatment administered naphthalin, which he prefers to naphthol. Nitrogenous food was also as far as possible eliminated from the dietary, and peptonised gruels given in place of custards. He gives details of four cases and a summary of his results in some thirty cases.

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[1] *Journal of Mental Science*, January, 1893.

Anæmia was prevented or removed to a very marked extent. Weight generally increased steadily. Pigmentation and dryness of the skin disappeared. Sleep was promoted and became natural and refreshing, and this occurred even when sulphonal had previously failed, and he thinks that the naphthalin suppresses the causes that prevent normal sleep.

Some mild melancholias were cut short, but more severe cases, though not shortened, were greatly improved, as "the mental distress and motor restlessness of melancholia rapidly disappeared, the suicidal cases became quieter, and the tendency to impulse in all the cases was almost entirely removed," and he claims for the treatment that it modifies the prominent troublesome and distressing symptoms of acute melancholia, and enables one to dispense with the use of sulphonal and hypnotics.

SINGER[1] finds that there is a distinct connection in time between excessive intestinal putrefaction and certain skin diseases, such as urticaria, acne vulgaris, and pruritus. That the two sets of troubles frequently appear together and may disappear spontaneously together, while treatment of the intestinal trouble has an undoubtedly beneficial influence on the skin trouble. He agrees with Herter and Macpherson in estimating the amount of intestinal putrefaction present from the quantity of aromatic sulphates passed in the urine, and he thinks that excessive intestinal putrefaction is not infrequently present without other obvious signs of dyspepsia.

While indican is often in excess in the urine in idiopathic urticaria, it is not met with in the erythema due to parasites, &c.

He has derived great advantage from the internal administration of menthol, which acts as an antiseptic, anti-fermentative, tonic and digestive, while purgation alone does no good.

He considers that the products of intestinal putrefaction are absorbed into the blood and come to the skin and act either on its vessels or its nerves.

Whatever the explanation, the skin troubles are connected with the digestive troubles, and give us a means of estimating their extent.

FREUND,[2] whose paper on "Autointoxikations-Erytheme" follows that of Singer, has apparently had very similar experiences. He has also seen the use of calomel produce good effects on the skin, but on the whole he prefers ol. menthæ. pip., which, in his hands has quickly caused indican to disappear from the urine, and rendered the stools free from odour.

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[1] *Wiener Klinische Wochenschrift*, Jan. 18, 1894.

[2] *Ibid.*, prev. ref. p. 39.

As to Herter's original researches on intestinal putrefaction in epilepsy, I have already said in BRAIN (spring and summer number, 1893, pp. 252 to 256) that he is in my opinion mistaking an effect for a cause, and that the uric acid, which, by its effects on contraction of arterioles and blood pressure deranges the cerebral circulation and produces the fits, interferes at the same time by contracting the arterioles and hindering the circulation, with gastro-intestinal digestion and allows putrefaction to take its place.

So that the fits and the intestinal putrefaction are two results of an excess of uric acid in the blood, and this accounts for their being seen together, and also for the excess of uric acid in the urine which Herter acknowledges is often found with them.

Exactly the same argument will apply to melancholia and Bright's disease, in both of which there is an excess of uric acid in the blood, causing that most common symptom in these diseases—high blood pressure.

Then, again, I have repeatedly pointed out that obstructed capillary circulation in the skin similarly produced, may account for numerous alterations in the function and nutrition of the skin from mere paleness and dryness, eruptions, &c., up to Raynaud's disease, atrophy and destruction; so that I can easily account for the relation observed by Singer between intestinal putrefaction and certain skin eruptions; here again we have two results of the same cause, and for several years past I have been treating diseases of the skin by clearing the blood of uric acid, and so improving its circulation.

The one thing common to all these conditions, call them neuroses or what you will, is obstructed capillary circulation and high-blood pressure, and, if I am right in my interpretation of the facts, it is common to them all, because it is the cause of all.

The obstructed circulation is in every way proportional to the amount of uric acid in the blood and urine, and is almost certainly due to an excess of this substance in the blood stream; all the other symptoms and phenomena are the result of this, and may consequently occur together, in various combinations.

Thus in migraine there is not only headache but also cold skin and extremities, scanty saliva and urine, fœtid motions, mental obscurity and depression, deficient excretion of water from the lungs, all these being results of obstructed capillary circulation, and this, again, is the result of an excess of uric acid in the blood, as is shown by the fact that all the circulatory troubles and symptoms are proportional to the excess of uric acid that is passing in the urine.

And the migraine and all its concomitant signs in the skin and extremities, glands, intestines, kidneys, brain and lungs, can be completely prevented by diminishing the intake and formation of uric acid, so that there is no excess in the body available for solution in the blood, and this can be done by diet, without the use of any drug whatever.

So that, while there is no doubt that these observers have been able to do good in these cases by attacking the intestinal condition, this is simply due to the fact that every improvement in digestion and absorption of food means a fall in the alkalinity of the blood and a diminution of the uric acid it can hold in solution, just as dyspepsia, however caused, means a rise in the alkalinity of the blood and an increase of the urate it holds in solution, so long as there is a supply of the substance in the body to be dissolved. In a word, the intestinal condition, though a result of excess of uric acid in the blood, tends, when it has come into existence, to keep up that excess, and make matters worse.

But none of these results prove that the epilepsy, headache, or melancholia on the one hand, or the skin troubles on the other, are due to the intestinal putrefaction. The only simple and complete explanation is that all these things are alike the result of one cause—the altered circulation which is common to all the organs of the body.

I can easily understand the good results obtained by Macpherson, for the calomel cleared the blood of uric acid, and the improving digestion tended to keep it clear, while the lowly nitrogenous diet introduced little or no fresh uric acid into the body. My researches on the causation of anæmia also completely explain both its presence and its rapid cure in his cases.

Herter's results with meat and milk are more difficult to understand, as meat introduces additional uric acid; and this had, apparently, been noticed by others, who failed to obtain good results with meat; but, as I have also pointed out, a dose of uric acid may cure a uric acid headache, because it raises the acidity for a time, and throws uric acid out of solution in the blood. Herter himself specially notices the plus excretion of uric acid in the urine that is frequently found with excessive intestinal putrefaction, but he has not had my experiences of the effects of uric acid on the circulation, and so he draws what I believe to be an erroneous conclusion from these researches, as well as from his original investigations on epilepsy, which were undertaken to test the truth of my statements as to the excretion of uric acid in that disease.

For further facts and cases bearing on these points I must refer those who may be interested to the second edition of my work on, "Uric Acid in the Causation of Disease,"[1] where I think they will find an explanation of the observed facts, accounting for the time relation between these disturbances, as well as for their connection with very numerous disease processes having a similar origin.

Since the above was written, Drs. Herter and Smith have published a further paper on excessive intestinal putrefaction,[2] and I am interested to see that in this they have much more to say about uric acid, and indeed acknowledge that it may cause several of the symptoms they are speaking of, quite apart from any excess of intestinal putrefaction, so that they are led to recommend the treatment of uric acid; and, though I cannot agree with their mode of treating it, I am glad to see that they are coming to acknowledge its power. I will merely point out, in conclusion, that the calomel, which several of these authors find so useful, does not act as a direct intestinal antiseptic; but, forming an insoluble compound with uric acid, it clears the blood of that substance, with the result that a free circulation with natural secretion and healthy digestion and absorption throughout the whole intestinal tract is again possible; and not the intestines only are affected, but every tissue throughout the whole body participates in the improved circulation and its results, so that you get diuresis diaphoresis, increase of saliva and of water given off from the lungs, as well as an improved cerebral circulation and consequent removal of the "neuroses"; and similar results follow the taking of calomel, whether given for excess of intestinal putrefaction or in more normal conditions.

A. HAIG.

Theodor Kaes on the Medullated Nerve-Fibres of the Cerebral Cortex. (*Archiv fur Psychiatrie*, xxv. Bd., 3 Heft, s. 695.)

This contribution concerns the comparative degrees of abundance of medullated nerve-fibres in different parts and layers of the human cerebral grey cortex, and in some measure also deals with these as influenced by age.

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[1] This is now out of print, but a third edition is in course of preparation.

[2] *New York Medical Journal*, June, 1895.

The results given in this paper are limited to those drawn from minute examination of the brains of two men, of a somewhat inferior class of society, dying of acute phthisis—one at the age of 18 and the other at that of 38.

Although brain-weights at different ages show that the growth of the brain, as indicated by weight, has nearly attained its completion by the age of 20, and that the weight remains about stationary from then until the age of 50, after which it declines, yet the development of the mental faculties and psychic activities does not occur in a parallel manner, since they stand at the height of their power from the age of 40 to 60. Instances of aged persons of particularly comprehensive and effective mental power show that the brain is still capable of active development in even the later years of life. But in microscopic measurement of separate parts of the brain and cortical layers, and in systematic critical estimation of the various cortical districts as concerns the abundance of medullated nerve-fibres in the several layers, we have a more subtle and more precise expedient than in simple weighing.

The examination of the two brains (aged 18 and 38 at death) showed that the formation and development of the cortical fibre-system advances much from the age of 18 to 38; and, moreover, that the formation and increase of fibres, and therewith the further augmentation of the sum total of the mental powers and of their effectiveness, is not yet exhausted at the latter age.

Between the ages of 18 and 38 the brain-convexity gyri increase considerably at the summit, this increase concerning chiefly the association-system of the second and third layers. On the other hand there is no increase (possibly even a lessening) of the lateral breadth of cortex at the brain-convexity. A general increase also occurs at the median and inferior surfaces of the brain, and, as a rule, equally distributed in the several layers, although there are some curious contrasts and contrary relative conditions on the two surfaces on comparing the first layer with the second and third.

Many interesting comparisons are given of fine cortical measurements, but these are too detailed for reproduction here.

As to the question: Which are the brain-districts richest in fibres?—while Vulpius believes the right anterior central gyrus to be so (in which he found the largest number of thick tangential fibres), and while Tuczek maintained that position for the central gyri, so rich in thick fibres, and for the occipital lobes with very numerous differently-thick fibres, Kaes indicates in a general way the motor cortical field (central gyri and back-end of frontal gyri), the temporal gyri (especially their posterior part), and the occipital, as being the districts which, at the age of 38, are the most advanced according to the criterion of fibre-value; whereas, at the age of 18, the temporal lobe is far behind the other two great regions in development of nerve-fibres.

The paracentral region possesses the greatest abundance of large fibres; the occipital outstrips all other cortical regions in the number of fibres and the early development.

In the motor zone, Kaes believes that he has proved that the posterior paracentral region in its general development is almost as plenteously endowed with fibres at the age of 18 as at 38.

The district poorest in fibres is the cerebral convexity at the frontal tip, and nearly like it in this respect is the gyrus rectus.

W. Julius Mickle.

Sur les connexions du ruban de Reil avec la corticalité cérébrale.—Dr. and Madame Dejerine. Société de Biologie, April 6, 1895.

After describing the various views as to the distribution of the fibres of the fillet (*ruban de Reil*), the authors refer to three cases of its ascending degeneration, in which the degeneration could be traced as far as the anterior corpora quadrigemina and the lower part of the optic thalamus, but no further. They found the posterior segment of the internal capsule intact, as well as the ansa lenticularis, the body of Luys, the globus pallidus, and the commissure of Meynert. These results confirm the experimental researches of Vejas, Singer and Munzer, and of Mott. Dr. and Mme. Dejerine also find descending changes in the fillet in old-standing lesions, involving the lower and posterior part of the optic thalamus and the fibres lying behind and external to the body of Luys; but in all these cases there was simply a slow atrophy, not a degeneration in the proper sense of the term, although it may lead ultimately to the complete disappearance of the fibres. This atrophy diminishes from above downwards, from the thalamus towards the nucleus of the columns of Goll and of Burdach, and is proportioned to the degree of destruction of the fillet and to the time that has elapsed since the original lesion.

In a case of a lesion in the sub-thalamic region which had lasted for twenty years, the atrophy, well-marked in the tegmentum of the cerebral peduncle and in the upper part of the pons, was much less marked at the lower border of the pons, while in the medulla, the inter-olivary stratum and the internal arcuate fibres of the opposite side were only slightly affected. The authors believe that the atrophy spreads slowly from the periphery of the neuron towards its cells of origin, and is analogous to that produced in the central segment of a bundle of fibres in the brain or medulla which has been divided by a lesion. In other words, they follow Monakow's view that the fillet is mainly composed of fibres which arise from cells in the nuclei of the columns of Burdach and Goll. They do not think it probable that any cells of origin are situated in the thalamus. When the lesion is in the pons, the descending atrophy is much more marked than when the lesion is in any situation above the pons. This depends not only on the fact that the destruction of the fillet is often more complete in these cases, but also that the seat of the lesion is nearer the cells of origin. They further agree with Monakow and Mahaim, in opposition to Flechsig and Hösel, in the view that the fillet is only indirectly connected with the cortex of the cerebrum. In nineteen cases of degeneration of the cerebral cortex of the Rolandic region and the parietal lobe, they found no case of degeneration in the fillet. At the most there was a slight diminution of this system due to a slight diminution in the size and not in the numbers of the fibres. They have also examined three cases of very old lesions which, besides affecting the cortex, had also destroyed the insula, the operculum, the putamen, the caudate nucleus and the globus pallidus, leaving, however, the internal capsule and the optic thalamus absolutely intact. In these cases there was absolutely no effect on the fillet. They conclude, therefore, that there is no direct connection between the nuclei of Goll and Burdach and the cerebral cortex, and that the sensory bulbo-cortical tract comprises two neurons, an inferior or bulbo-thalamic neuron, represented by the median fillet, and a superior or cerebral neuron, connecting the thalamus with the cerebral cortex.

ALEXANDER BRUCE.

Putnam on the Relation of Infectious Processes to Diseases of the Nervous System. Pathology and Etiology.

(*Am. Jour. Med. Science*, 1895, p. 254.)

(1) The author inquires into the pathogenesis of the nervous affections which are obviously of infectious origin; (2) investigates the claims of the affections which are only suspected to be of infectious origin; (3) investigates the contributive causes which make certain parts of the nervous system peculiarly liable to suffer from infectious influences; and remarks that the direct effects of infection are not to be feared more than the prostrating influence of the original struggle.

Out of 5,000 cases of affections of the nervous system he found that about 500 had had, within a year before the onset of the illness, some infectious disease. About 80 per cent. of these affections were post-influenzal and post-syphilitic (even excluding the cases of paretic dementia and ataxic tabes with a syphilitic history, which numbered respectively 29 and 71); the rest, in order of frequency, were cases preceded by diphtheria (53), typhoid (47), scarlatina (14), malaria (14), measles (9), gonorrhœa (8).

In order to consider, first of all, to what extent the results could be explained without referring them to the direct action of the infective agent, an intimate acquaintance with many influences, of which the following are the chief, is required:

(1) The nervous system has certain natural proclivities to certain forms of disease in the production of which the infectious process only joins hands with developmental or inherited tendencies, or accentuates the habitual strains of life [laws of "devolution" (Jackson); laws of "degeneration"; disproportion between waste and repair (Edinger)]. Our knowledge of these tendencies is still far from complete.

It is not only the typical psychoses and neuroses which can arise in this way, but also some of the acute and chronic degenerative affections, such as symmetrical polyneuritis and the different forms of tabes. These factors seem, however, to be called out more strongly by certain infectious processes than by others, and made to operate in more or less definite ways, so that, clinically, the connection between the infection and the final disease, though indirect, may be close.

(2) The infectious processes may give rise to secondary disorders of nutrition, of cardiac, vascular, digestive, sclerotic and metabolic origin, and a primary infection is liable to become

complicated at any part of its course by a secondary infection with its own train of consequences. Poisonous substances are being formed in, and constantly eliminated from, the body; when this elimination is checked or arrested, as often happens in the course of infectious processes, an auto-intoxication takes place which bears its share in the production of symptoms [Bouchard (16)]. Such researches as these should make us cautious as to how to explain the clinical and anatomical effects of infection. Bouchard comes to the conclusion that there are two classes of influences at work in cholera infection: first, the poison primarily produced by the specific organism; second, a secondary poisoning due to uræmia (itself a complex and variable cause), and that each of them have their own symptoms, which may succeed or alternate with each other.

The principal effects produced by the infective agents on the nervous system are:

A. A series of *primary disorders* is induced, not directly due to gross lesions, but to the toxic action of one or more of the *soluble elements* of the (often complex) poison.

In this class of primary toxic affections belong the symptom-complexes of tetanus, rabies and whooping-cough, a portion of the cerebral, spinal and meningeal symptoms of acute infectious diseases, and perhaps many of the characteristic symptoms of diphtheria, Landry's paralysis and chorea. Symptoms of peripheral nerve disorders *may* be due to poisoning without recognisable lesions. Ether is well known to be a nerve poison, and if injected into the tissues in the neighbourhood of a nerve, degeneration will follow. On the other hand, when an exposed frog's nerve is kept moistened at one point with ether, its power of conduction may be inhibited for at least forty minutes at a time (Bowditch), and yet on the removal of the ether the nerve may resume its functions as if uninjured.

The signs characteristic of the nervous affections following infectious processes are due far more to the toxic substances which are developed in the course of bacterial action than to the micro-organisms themselves.[1] The formation of tubercle and abscess, and the exudation of purulent meningitis imply the presence of micro-organisms, and bacteria are found now and then in the brain, spinal cord and nerves, under various circumstances, without its being possible to say as yet what their relation is to the lesions accompanying them.

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[1] See *Therapeutic Gazette*, 1894, p. 185; also Flexner (Toxalbumins, &c.), *Medical News*, August 4, 1894.

It is only in the case of lepra that the bacteria may be said actually to invade the nerve-cell or nerve-fibre (Babes [9] and others), and even here the occurrence is not constant (Kuhne, Zambaco-Pacha). Ordinarily, the bacteria lie in the lymph spaces. The presence of histological cell-changes after infection does not necessarily imply the direct action of bacteria, for they may occur in a typical form where only sterilised and filtered cultures have been used, as has been shown by Welch (93) for the case of diphtheria. (See also Flexner, *loc cit.*)

B. The *peripheral nerves* suffer from disorders of nutrition due to—

(1) Infiltration with toxic substances; (2) Interstitial and vascular disorders; (3) Diseases of their trophic centres. As compared with neuritis due to mineral poisons, the infectious neuritis is perhaps more frequently due to the second of these causes (Cramer), but the distinction is not absolute.

C. The *cerebral* and to a less extent the *spinal membranes* become breeding-places for colonies of bacteria. Possibly meningitis is occasionally due to the irritation of soluble toxines, as in some cases of scarlet fever [Bendel] (*Wiener med. Presse,* 1894, No. 23).

D. Focal (generally hæmorrhagic) non-suppurative *lesions of the brain, cord,* or *nerves* may be set up by either bacteria or their toxic products. We know as yet but little of this matter except through experiments with animals, though acute lesions of this sort are met with which are supposed to be of infectious origin. Bacteria of many kinds have indeed been found in the central nervous system, though rarely in man, as the obvious cause of focal inflammation, with the important exception of abscess and tubercle. Characteristic bacteria are also claimed by Pfuhl to have been found in the foci of encephalitis following influenza. We now know that specific toxines are capable of exciting focal lesions, and we are looking out for clinical evidence indicating a direct causal connection between the soluble poisons of influenza, diphtheria, variola, scarlatina, typhoid, gonorrhœa (Leyden, Hayden and Parmentier, Dufour), and the focal myelitis which sometimes follows them.

Enriquez and Hallion (*Soc. de Biologie,* April 14, 1894) have also found that the toxines of diphtheria injected under the skin of dogs may cause, *if the dose is not too large,* intense congestion of the grey matter of the cord, with hæmorrhages, and foci of inflammation characterised by formative irritation of the neuroglia and destruction of nerve elements. Symptoms suggesting a similar process were also seen in an ape. Animals are, perhaps, more subject than man to *toxine myelitis*. At any rate, localised destructions of the grey matter of the cord have been found by Gilbert and Leon (*Semaine Méd.*, 1892, p. 65), and more recently by Thoinot and Masselin (*Rev. de Méd.*, June, 1894), after inoculation with virulent cultures of the bacillus coli communis; by Bourges (*Soc. de Biologie*, 1893) after injections of erysipelas cultures (streptococcus pyogenes ?); and by Vincent (*Arch. de Méd. expérimentale*, 1893), after inoculation with typhoid bacillus. Roger, also (*Ann. de l'Institut Pasteur*, 1892), has produced a poliomyelitis by inoculation of rabbits with a streptococcus obtained from erysipelas, and his observations are particularly interesting from the fact that this result only attended a certain phase of virulence of the organism, which he was not able to reproduce at will.

It is probable that acute myelitis in man is sometimes of infectious origin. The possibility of secondary infection should, of course, never be lost sight of. Auché (*Semaine Méd.*, Nov., 1894, *Soc. de Biologie*) found a secondary streptococcus infection to be the probable cause of a myelitis following variola.

E. Diffuse lesions of nutrition of nerve-cells and tracts are being found more widely in proportion as our means of investigation improve, as witness the changes in the posterior columns observed by Bikeles in diphtheria; many of these may, however, be due to œdema or disorders of circulation (see Edinger, *Sammlung klinischer Vorträge*, No. 106).

The earliest lesions in cases where none were supposed to be found are described by Golgi (41) as occurring in *rabies*. The nerve cell shows impairment of its nutrition by peculiar changes affecting the size and contents of the nucleus. The cell-protoplasm, the endothelium of the vessels, and the neuroglia take part in the change, which is one first of irritation, then of reaction and repair. He denotes the whole process an "encephalo-myelitis parenchymatosa." The irritative stage is associated with active hyperæmia. He asserts that the histological peculiarities are not pathognomonic. They only mark where the poison has struck, and perhaps indicate the severity of the blow. It is especially noteworthy that most of these changes, as observed in rabbits, were found before the stage of complete paralysis had been reached, and similar lesions were in fact noted in the cerebellar cortex, within four or five days after inoculation and before any symptoms had appeared. One is reminded by this statement of the peculiar form of parenchymatous neuritis observed by Gombault in guinea-pigs, as a result of chronic lead-poisoning, at a period when the animals appeared to be perfectly healthy. Golgi further notes that these changes are very similar to those which he had discovered in chorea.

Minute changes such as Golgi describes are only to be accepted as pathological when made by trained observers and under the most favourable conditions of preparation. Even then it might not always be easy to say that we were not dealing with the effects of fatigue or of œdema, and still more difficult to distinguish between the action of specific toxines and that of the toxic products of disordered nutrition. A good deal has been made of the toxic origin of vacuolation of the nerve cell [see Campbell (20)]. Vacuolation has, however, been noted under such varying conditions that one must wait to see whether it indicates anything more than one of the ways in which nerve cells regularly degenerate. It certainly affords no means of distinguishing between bacterial poisons and others (Afanassijew,[1] Campbell).

The distribution of the lesions which are noted by Golgi is more important. There would have been no special reason to suspect that the brain and cerebellum as well as the medulla and spinal cord would be involved in rabies, and the fact that this is found to be so, assuming that the observation is correct, is an additional reason for thinking that many poisons which seem to be very limited in their action, are really universal nerve poisons, and will excite widespread lesions if time enough is given them.

When we pass beyond these minute parenchymatous lesions, the change which seems most characteristic is vascular congestion with infiltration of leucocytes, often associated with hæmorrhage, and perhaps due to endothelial changes, at least in part. This tendency has been often discussed elsewhere; but the œdema so produced may in itself be a cause of some of the associated nerve-lesions. It might cause degeneration of nerve-fibres, by pressure or disturbance of circulation, and such destruction of nerve cells as is seen in poliomyelitis. When the hæmorrhage and exudation are of infectious or toxic origin there is probably infiltration of poison into the nerve elements and the other tissues in the immediate neighbourhood of the affected vessels; but whether this infiltration precedes the congestion, or is a necessary factor in its production and localisation, is unknown. (See Flexner, *Med. News*, Aug. 4, 1894, on similar affections of other organs.)

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[1] "Acuter u. chron. Alcoholismus," *Beitrage zür Path. Anat. u. Allgem Pathol.*, 1890, p. 443.

The next question is with regard to the influences which increase the liability of the nervous system to suffer from the infectious processes—the contributive causes of disease.

The question is divisible into two parts. The first concerns the influences which lessen the power of resistance of the body as a whole to invasion by the organisms of disease, the second concerns only the influences which increase the vulnerability of the nervous system to the poisons produced by the organisms.

The distinction is an important one. A healthy person, with intact mucous membranes and vigorous nervous centres (see Bouchard, Chorrin, Gamaleia, Buchner, Gley), might resist the onset of infection, while even a healthy nervous system might not be able to withstand the action of a specific virus. Indeed it is open to question whether the poisons do not sometimes affect healthy more strongly than enfeebled nervous centres, for the response to the action of a poison (*e.g.*, that of tetanus) is often in reality a sort of reaction which may imply a normal vigour. A difference is also likely to exist between the character and degree of the functional response to acute intoxication, and the liability on the part of the nervous system to suffer local lesions of nutrition, which experiment has shown to attend toxine poisoning as well as the direct action of bacteria.

Strong nervous centres are probably as liable as weak ones to exhibit the *toxic symptoms* of acute infectious poisoning, while feeble ones are more prone to show *disorders of nutrition* under the added strain of the presence of the poison, especially if this is reinforced by impaired circulation or œdema.

Among the post-infection disorders of the nervous system there is none so common as *neuritis*, usually, though not always, symmetrical. The infectious toxines are capable of causing neuritis, but we probably include under this name a number of other affections. Such are: (1) the neuritis attending the first stage of an infectious process before there has been time for the development of general disorders of nutrition; (2) the post-infectious neuritis, in the production of which general disorders of nutrition and a variety of other causes play an important part; (3) the acute, usually fatal and hæmorrhagic, neuritis of unknown origin. Nor should it be forgotten that what we have considered as neuritis is often associated with nuclear and diffuse spinal lesions. Paralysis of the peripheral type may be unattended by demonstrable lesions, and this fact has lent support to the view that the spreading forms of paralysis (Landry) may belong to the same category.

According to Ross[1], Leyden[2] and others, there is no marked difference in the clinical history of some cases of spreading neuritis and Landry's disease, and the mere absence of demonstrable lesions does not justify us in setting apart the latter cases as belonging to a special group. But other pathologists (Hun[3]) hold that so long as there does appear to be a class of cases where the disease is generally fatal, and yet no definite lesions are found, there is nothing gained by lumping them, for the sake of pathological unity, with demonstrable cases of neuritis. Putnam thinks that the mere absence of demonstrable lesions in a given case of poisoning cannot be taken as proof that the pathogenesis of the symptoms is not the same as in another case where lesions were found. That which is characteristic is often the primary action of the poison rather than the effects of the lesions, even when lesions are present. The history of tetanus and diphtheria, and perhaps rabies, gives warrant for this assertion, for in some instances of these diseases few or no lesions are discoverable, while in others the lesions are more extensive than the symptoms would have suggested. Goldscheider (*Berl. klin. Wochenschr.*, June 4, 1894, p. 546) takes the position that the presence or absence of demonstrable lesions in Landry's paralysis is a matter of slight importance. Jolly (*ibid.*) takes the position assumed by Hun.

Part of the symptoms of acute hæmorrhagic (infectious?) neuritis are unquestionably of peripheral origin—those, namely, which are due to the pressure of the endoneural exudation. It is, however, far from certain whether death, in these cases, is due to the neuritis or to a poisoning of the nerve centres.

Leyden and others have noticed that in some of these cases changes in the spinal cord are discoverable (swelling of axis-cylinders). The author described such a case where death occurred at the end of a week from respiratory paralysis (*New York Journal of Nervous and Mental Diseases*, 1887). Edinger (*loc. cit.*) thinks that this swelling of axis-cylinders is often referable to œdema, but it is probable that it at least implies an impairment of resistive power of morbid origin.

The more chronic forms of neuritis which follow known infection are probably toxic in origin; but it is uncertain whether the poison is identical with that which caused the primary infection, or is due to secondary disorders of metabolism; the stimulants given during the illness may be a contributive cause (Starr).

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[1] "A Treatise on Peripheral Neuritis," 1893.

[2] *Zeitschr. für klin. Med.*, vol. xxiv.

[3] *New York Medical Journal*, May 30, 1891.

The peripheral nerves (or their trophic centres) are singularly sensitive to poisons of many kinds. Ross enumerates forty causes of neuritis, and among those recently noted are puerperal conditions [Moebius (62), Bassette (11), see also *Nouvelles Arch. d'Obstet. et de Gynécol.*, 1894, No. 9,] erysipelas [Grasset, Lew,] septicæmia [Lloyd and Riesman (58)], pyelonephritis [Dana], suppuration of the pleura [Fiessenger (35)], gonorrhœa [Engel-Reimers, *Jahrbücher der Hamburger Staatskranken Anstallen*, 1890].

The lesion is not precisely the same in all these cases. After acute infectious diseases the morbid process may centre around the blood-vessels, and is said to be rather interstitial than parenchymatous [Cramer (22)]. The spinal cord is apt to be involved with the nerves in these cases. In some instances the parenchymatous degeneration may be due to the pressure of œdema. In the acute (hæmorrhagic) form, which is supposed to be of primary infectious origin, the trunks of the nerves and the nerve-roots may be involved, as well as the peripheral portion. Different varieties of degenerative neuritis are also described, such as the "periaxillary" and "segmental" forms (see Cramer). Research may show that where an irritant organic poison is circulating in the blood, capable of causing neuritis, the endothelium of the vessels will show changes like those described by Golgi as occurring in rabies.

Cerebro-spinal membranes.—The symptoms are probably due partly to the exudation itself, partly to the irritation of the sensitive membrane, partly to the absorption of toxic products. The organism of both the epidemic cerebro-spinal meningitis—the sporadic forms not associated with suppuration elsewhere—and the meningitis which sometimes accompanies pneumonia, is the so-called pneumococcus, or micrococcus lanceolatus. The history of this important bacillus has already a voluminous literature, but interesting analyses are given in the papers by Netter (63), Welch (93), Foà (*Zeitschr. für Hygiène*, 1888), and in the research by Flexner and Barker (37) into the nature and causes of a recent epidemic of cerebro-spinal meningitis. This (pneumococcus) form of meningitis seems to be by far the most common variety even of those occurring independently of epidemics of pneumonia. Out of thirty such cases Netter found sixteen to be due to this organism, while the largest number induced by any other single organism was six. Out of forty-five cases of primary meningitis reported by other observers and collected by Netter, thirty were instances of pneumococcus infection.

Flexner and Barker think it more probable that the micrococcus passes into the digestive tract and thence, by way of the blood, to the brain; the injection of cultures does not excite meningitis in animals unless made directly into the cerebral cavity.

The high importance of inflammations of the antrum, nasal sinuses, and ethmoid cells for the introduction of organisms to the cerebral membranes, has of late been commented on by many writers—Beck, Macewen, Dmochowski (29), Neumann and Schaeffer (64), Allport (4). (See Macewen's "Pyogenic Diseases of the Brain" for the mode of invasion of the organisms, the formation of abscess, and the origin and spreading of thrombosis and meningitis.) Meningitis may also be due to the bacillus of typhoid. (Tictine, Adenot, Drews.)

It is possible that the poison of scarlet fever and measles may occasionally be the direct cause of meningitis (see Bendel, *loc. cit.*); *acute, primary, hæmorrhagic encephalitis* occasionally follow various forms of the acute infectious disorders, and also occur without known infection, though perhaps not independently of a toxic cause, a possible source of error in the diagnosis and even in the pathological examination; the author reported a case of post-influenzal disease (75) which was believed to be an example of this disease, but subsequent examination showed that nothing more than œdema could be made out with certainty. An additional case is reported by Koenigsdorf (*Berlin. klin. Wochens.*, 1892, No. 9).

Relation of the *chronic spinal scleroses to infectious processes.*—We know how frequently locomotor ataxia and lateral and mixed scleroses follow syphilis. The relation of disseminated sclerosis to several infectious processes, and especially to malaria, is almost equally evident. We do not know what is the nature of this connection, but it clears the way to note again the natural proclivity of the nervous system to become so diseased. Thus we may admit other causes besides the specific virus. The case of syphilis in its relation to tabes, &c., is a case in point. It contradicts all that we know of the action of syphilis, and the usual seat of its lesions, to assume that it is the direct cause of these degenerative scleroses.

Gummatous inflammation may indeed give rise to many of the symptoms of tabes, and this may be cured; and it is true, also, that various modes of treatment may help ataxics for a time. But the relation of syphilis to locomotor ataxia is to be explained only on the assumption that some new (toxic ?) influence is brought into play, perhaps on a predisposed nervous system.

Sclerosis of the posterior columns occurs as a sign of structural weakness in Friedreich's disease;[1] it follows ergot, pellagra, and lathyrus poisoning; it occurs as a part of the degenerative process in chronic paralytic dementia; as a consequence of morphine-poisoning in dogs [Alt(5)]; and its outbreak is precipitated by such processes as influenza and trauma.[2]

Posterior sclerosis is sometimes associated with progressive pernicious anæmia, and perhaps with other affections which originate in auto-intoxication (from the digestive tract ?).

The first communication on this subject was by Lichtheim, in 1887 (*Sixth Congress für innere Med. zu Wiesbaden; Verhandlungen*, p. 84); but, in 1893, Dr. W. Minnich published a careful monograph on the subject, and gave full reports of all the cases observed up to that time. The author had previously observed a series of cases, one or two of which were certainly cases of pernicious anæmia, and in 1891 he published a paper under the title "A Group of Cases of Sclerosis of the Spinal Cord associated with Diffuse Collateral Degeneration, occurring in Enfeebled Persons past Middle Life, and especially in Women." (*New York Journal of Nervous and Mental Diseases*, February, 1891.) Various partial causes were noted, and among them lead-poisoning and chronic diarrhœa.

It is true that Minnich declines to admit that his cases are of the same order as tabes, or ergotism (Tuczec; Grünfeld, *Arch. für Psych. u. Nervenkrankheiten*, vol. xxi., p. 618); but he is unable to explain them on a different basis except that they seemed to originate in foci of disease related to the vessels, and he admits them to be probably toxic in character.

His interesting paper also contains an account of another form of change in the posterior columns which seems to occur in some cases of pernicious anæmia, probably toward the end of life, and to be due to œdema. The rest of the cord is affected to some extent [*cf. Bikeles* (95)].

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[1] What is true of posterior sclerosis is true, also, of the other spinal scleroses and degenerations and the mixed forms, among which latter Friedreich's disease belongs. (See a paper and discussion by Strumpell and others. *Wiener klin. Woch.*, 1894, No. 4, p. 69. Mendel here remarks that even multiple sclerosis may rest on a hereditary basis.)

[2] Cf. Morton Prince, *Neurol. Assoc.*, 1894, and Klemperer, *Zeitschr. für klin. Med.* xvii., 1890. The interesting observations and ingenious theory by Obersteiner and Redlich (95) does not close the discussion as to the pathogenesis of tabes. The argument that the posterior roots are squeezed at their entrance into the cord by a thickening of the membranes, of syphilitic origin, fails, as the authors recognise, to account for many other associated lesions of tabes, and to recognise the rapid increase in symptoms which attends general depressive influence.

Lichtheim refers to other conditions besides those here mentioned, which contribute to disease of these tracts, and suggests the effect of sensory excitations.

Edinger's theory is that repair does not keep pace with waste. (*Sammlung klinische Vorträge*, No. 106.)

It is true that just as there are differences between the different forms of neuritis, so, also, the posterior sclerosis which follows syphilis is probably not identical with that due to the other causes mentioned. The ergot-sclerosis, for example, is not so malignantly progressive, and is, perhaps, anatomically different; and there are other peculiarities of the different varieties, some of which are pointed out by Raymond (78) and by Friedheim (38). But there is a strong bond of resemblance connecting them all. There is even some evidence, apart from the important case of Friedreich's disease, that tabes may be a member of the so-called neuropathic family (especially when it develops in the later years of life).

Among the less common lesions of syphilis the author mentions a form of myelitis closely resembling the ordinary transverse dorsal myelitis. The disease is of slow development, and the spastic element is strongly marked, in proportion to the paralysis and the loss of sensibility, which are relatively slight. The patients may improve, especially under the influence of an energetic specific treatment, while, at the same time neither the symptoms nor the improvement point to typical gummatous disease. This form was first described by Erb, in the *Neurologische Centralblatt*, No. 6, 1892, under the heading of "Syphilitic Spinal Paralysis." (See also Kowalewsky, *Neurologisches Centralblatt*, 1893, No. 12, and Kuh, *Deutsche Zeitschrift für Nervenheilkunde*, 1893, iii. 6.)

Another form of syphilitic disease is that where more or less typical lesions of syphilis are associated with those of tabes: important, because cases of this sort are perhaps those where specific treatment has some results. Dinkler (*Deutsche Zeitschrift für Nervenheilkunde*, 1893, iii. 4 and 5) reports a striking case of this kind, and gives references to the earlier literature. (See also Sachs, *New York Med. Journ.*, January 6, 1894.)

Diphtheria.—Conflicting views respecting the nerve lesions are held by Gowers, Arnheim, Hochhaus, and Cramer. Gowers and Arnheim describe the neuritis as parenchymatous, while

Hochhaus and Cramer think that in diphtheria (as in the other infectious processes) the primary change is vascular and interstitial, and Hochhaus refers many of the symptoms to pressure of œdema. Gowers quotes an interesting observation by Boissarie (*Gaz. Hebd.*, 1881, Nos. 20 and 21), describing a series of cases of paralysis of the palate, eyes, limbs, heart, "perfectly like that which occurs after diphtheria, and accompanied by albuminuria. The remarkable fact is that in these cases of primary palsy there was no history of preceding sore-throat, and in several of the cases distinct diphtheria followed the paralysis, which lessened during the throat affection. Some of these cases of primary palsy seem to arise by infection." At the same time there occurred a series of cases of severe diphtheria. We may now assume that careful examination of the throat would have shown that the bacteria were really present, even where no soreness of the throat had occurred.

The experimental study of the nerve lesions of *diphtheria* (see Gowers, 2nd ed.) has given conflicting results. Good observers have repeatedly failed to discover histological changes to account for paralyses experimentally induced, and although changes have been found in man, especially in the peripheral nerves, yet they have been occasionally missed (even by recent observers; see Bikele's paper in *Obersteiner's Arbeiten*, Heft 2, 1894), and perhaps represent only a terminal stage in the process.

Quite recently an experimental research by Stscherbak (86) has given more positive results. The changes which he observed can be divided into (1) those involving the peripheral nerves, consisting in well-marked degeneration, which on the whole corresponded in position and intensity to the paralytic symptoms, and (2) those involving the spinal cord, membranes, nerve-roots, and the muscles. The former he regards as constant; the latter rather as incidental than typical.

The diseases of the nervous system which are suspected, but not proved, to be of infectious origin, are, especially, beri-beri, poliomyelitis, Landry's disease, certain forms of myositis, neuritis, myelitis, some of the cerebral palsies of children, chorea, disseminated sclerosis and other cerebro-spinal and spinal scleroses, amputation neuritis, herpes zoster.

The grounds on which a disease may be suspected to be of infectious origin are, when bacteria are found in the tissues; when the outbreaks occur in epidemics or are related to seasons or locality; when vascular and histological changes characteristic of the infection are found; or specific toxicity of the blood and urine; or if the disease has become much less frequent since the introduction of antiseptics.

On this latter ground the intractable *neuritis following amputation* has been suspected to be of infectious origin [Page (70)]. It is true that even when great care is taken to prevent infection, amputation neuritis still occasionally occurs, but it should be remembered that at the best surgical asepsis can be but relatively perfect.

As regards the lesions to which the nervous system is liable as the result of infectious disease, there are some which are more or less characteristic in view of its structure and liabilities, and others which it shares with other organs and tissues of the body. Of the latter class are the vascular lesions, hæmorrhage and exudation, which are of vaso-motor origin or due to changes in the vascular walls such as are liable to occur in any part of the body. Where well-marked evidences of these vascular lesions occur it is common to consider them not only as evidence of infection, but also as the cause, rather than the consequence, of the parenchymatous lesions with which they are associated. Goldscheider (40) supports the theory of the infectious origin of acute hæmorrhagic neuritis or myeloneuritis and encephalitis, as well as of *poliomyelitis*. In the latter disease the signs of congestion and leucocyte infiltration are prominent, and a case published by Putnam in the *Journal of Nervous and Mental Diseases*, 1883, not included in Goldscheider's (40) or Siemerling's (85) lists, agrees with this view. The author expressly notes that the wasting of the ganglion cells was not by groups, but "depended on their position with regard to the centre of inflammation," and that "the process is not one which affects ganglion cells materially more than other tissues in their neighbourhood." He suggests "that the original morbid change was one affecting the vessels and the circulation."

Another argument in favour of the toxic theory of poliomyelitis is that this disease is certainly related to the acute (often hæmorrhagic) polyneuritis, and that for this disease we find still more evidence of toxic origin. In both affections we see the strong vascular element in the onset; in both the occasional relation to acute infectious disease, and with neuritis the relation to beri-beri.

As regards the analogy between poliomyelitis and neuritis, several instances of the combined occurrence of the two diseases have been published [Gowers (43)]. In Putnam's case of central myelitis the clinical evidences of severe and widespread peripheral neuritis were unequivocal.

"Poliomyelitis" may include several affections which ought to be separated; or, in other words, that several different poisons are liable to affect the anterior horns of the spinal cord, exciting results which, though in the main alike, may differ in detail.

The experimental evidence is in favour of this view, as well as the varying characters of the so-called epidemics of poliomyelitis. (See especially Caverly, *Medical Record*, Dec. 1, 1894, and Macphail, *Medical News*, Dec. 8 and 22, 1894). The number of cases studied was 126, and the symptoms of some of them were so peculiar that, although the diagnosis of "poliomyelitis" was considered as the most probable one, it was not regarded as perfectly satisfactory. [See also Siemerling (85)].

As regards the infectious origin of *Landry's paralysis*, all we can say is that it occasionally follows recognised infectious diseases, and that its abrupt onset and rapid course, often at least unattended by gross lesions, point definitely to some toxic cause. There is no reason at present for calling this cause bacterial, any more than poliomyelitis.

The theory of the infectious nature of *chorea* is interesting, but still far from being proved. The subject is well presented in papers by Pianese (73); Dana (24); Berkley (*Johns Hopkins Reports*, 1891), and others.

Pianese's investigation dates back to 1891. The only point which needs to be mentioned here is that he found in the cervical portion of the spinal cord, the medulla oblongata, and the cerebellum—but not, it would seem, in the brain proper—a bacillus which he considers as specific, and which he was able to cultivate and use for experimentation. Injections of pure cultures into animals gave rise to motor symptoms and often death, but apparently did not cause anything that could be called chorea. In the animals experimented with, the bacillus was found again in various parts of the nervous system, including the brain. Pianese has also examined the blood in a number of cases of ordinary chorea, and in one of them succeeded in finding this bacillus.

Dana's (24) first article was published in BRAIN, in 1890, and contained a report of accurate investigations into the pathological anatomy of chorea. His examination confirmed and defined those of previous observers (Hanford, BRAIN, 1889-90, p. 129). It seems clear that the most obvious change consists in vascular congestion, dilatation, sometimes leading to thrombosis or capillary hæmorrhage, and that widespread degeneration of the nerve elements may also occur, besides subacute meningitis. In Dana's (26) second paper, besides changes of these sorts (though in this case the signs of vascular congestion were less marked), a small number of micro-organisms resembling the diplococcus lanceolatus were found in the deeper layers of the pia mater and the superficial part of the cortex. The case was not one of typical chorea, however, and the number of micro-organisms was to few too warrant their being considered other than incidental.

Berkley describes a series of minute changes, chiefly in the membranes and vessels. Areas of focal inflammation were found in various parts of the brain, medulla, and pons, and among other conditions which are important as indicating the infectious character of the disease, there was a peculiar endarteritis, which, as Berkley says, is rare in persons of twenty-seven, except from an infectious cause. Micro-organisms were not found, but deposits of detritus were observed, which resembled the remains of bacteria. In the second part of his paper, which describes the case of a choreic dog, Berkley compares, in adjacent columns, the changes which he found with those given by Babes (8) as characteristic of diphtheria, in order to show the analogy between the two processes.

If ordinary chorea turns out to be of infectious origin we must suppose that the bacillus which causes it is present in an inactive state in large numbers of persons, for the disease may develop instantly, in consequence of fright, and some sort of predisposition must be present, for the disease is one which occurs chiefly at certain seasons of the year. We cannot say whether the lesions point to a toxic agent due to bacterial action or to a toxic agent induced by some form of disordered metabolism, or due to retained excretions.

With regard to the other affections, the argument in favour of the infectious origin is mainly that they are apt to follow other general diseases admitted to be infectious, like the acute exanthemata, but the exact significance of that argument is uncertain. One or two recent observations suggesting a relation between malaria and *disseminated sclerosis* have been published (Marie, Torti and Angelini), and Putnam has seen a striking example of the sort. A man in middle life and of good health was attacked suddenly with characteristic and progressive intention-tremor and scanning speech immediately after a sharp malarial attack. Under treatment by arsenic there was a slight improvement only.

The argument with regard to *herpes zoster* consists essentially in its tendency to occur in epidemics—though it must be admitted that this tendency is by no means strongly marked—and in a somewhat marked liability to follow other acute infectious diseases. [See Weigert (92), Schäffer (81), Kaposi (51), Wasielewski (90)].

The infectious theory of *epilepsy* calls for critical examination. The argument that the attacks are temporarily arrested by some intercurrent infectious malady, which might be supposed to furnish antitoxines, to the blood, cannot be seriously advanced by anyone familiar with the history and therapeutics of epilepsy [see Féré (33), Marie (60), Quériand (76), Séglas (84), Lannois (55)].

The origin of *beri-beri* is still a matter of doubt; some recent observations (Scheube, Glogner; *Virch. Arch.*, vol. cxxxii. p. 50), suggest that it is a protozoal disease, analogous to malaria. Glogner calls attention to the periodicity of the outbreaks of the symptoms, and claims to have found changes in the blood and an organism resembling the plasmodium. (Some recent observers attribute the disease primarily to imperfect hygiene, like scorbutus.)

Another means by which it is thought that the infectious character of these nervous diseases may be tested is the chemical and physiological study of the blood and urine. [Albu (2), Séglas, Fisichella, Ewald, Bouchard, D'Abundo (27), Régis and Chevalier-Lavanol (79), Voison and Peron (89). See also *Boston Medical and Surgical Journal*, September 14, 1893, and *Archives de Neurologie*, 1893, p. 240.] It is evident that before much can be done here, the toxicology of normal urine must be thoroughly understood, and for this reason the recent publications of Bouchard are of decided value, though the conclusion which he reaches will doubtless require modification. Bouchard maintains that if men are not poisoned by the products of their own tissue-metamorphosis, it is because the poisons which are constantly being formed are as constantly carried off from the blood in the urine. He finds seven toxic substances in normal urine, each of which has properties of its own. The first is diuretic in its action, and consists practically of urea. This is only toxic in a considerable dose, and is in general prophylactic in its tendency, since by increasing the quantity of urine it helps to carry off the other substances. The second ingredient is narcotic in character, and its chemical nature is not known, except that it is soluble in alcohol. The third is also soluble in alcohol, and has the peculiarity of causing salivation. The other substances are characterised by their power of causing convulsions, contraction of the pupil, reduction of heat. One of the convulsive agents is potash. In uræmia, &c., all these poisons are increased in quantity.

SUMMARY.

It is important to separate (*a*) the symptoms and lesions which we can assert to be directly due to the specific toxines of the various infectious diseases, from (*b*) the effects of anæmia, œdema, intoxication, from disordered metabolism, on the one hand, and on the other hand from (*c*) the affections which spring into existence because the nervous system was prone to them, either from peculiarities of structure, anatomical surroundings, circulation, and the like; or because other causes had been at work, to which the infective poison only contributed new effectiveness for evil.

In the first class (*a*) we can fairly place the symptom-groups of tetanus and rabies; and with reasonable probability the chill, pains, paræsthesias, acute delirium, disorders of micturition, and other symptoms which characterise the outbreak of many acute febrile disorders, though it must be admitted that we know little or nothing positive about their pathogenesis.

The focal hæmorrhagic encephalitis and myelitis occasionally observed after influenza and other infectious processes is probably due to the action of the specific poison on the tissues and the vessels. Acute poliomyelitis and acute hæmorrhagic neuritis may be of the same origin, though these opinions need further substantiation.

As regards tetanus and rabies, it is not probable that the anatomical lesions which have been found after death (Golgi and others) are actually the anatomical substratum of the characteristic symptoms. They may have been simply of correlative origin.

Post-infection meningitis is generally of purulent character, and implies the presence of micro-organisms; but the non-purulent form bears a relation to infectious disease which is in need of further study.

The paralyses of diphtheria are probably attributable to the direct action of the poison, but, as in case of tetanus and rabies, it is by no means certain that the lesions sometimes found (though often missed) are the true cause of the symptoms. It is more probable that the first and characteristic effect of the poison is to excite a chemical or molecular change, and that this alone is strictly characteristic. Sometimes the paralysis passes away too rapidly to be attributed to neuritis. The lesions, when present, may be many and various, and the neuritis, which is the commonest form, is of varying types. (Meyer, *Virchow's Archiv.*, 1881, vol. lxxxv. p. 181.)

The lesions met with in meningitis, and in a certain sense those characteristic of tuberculosis and syphilis, are directly due to bacterial action.

In the following cases the influence of cause *a* is more or less combined with those of causes *b* and *c*.

The question as to the origin of the post-infectious multiple neuritis is one of great interest. The most obvious position to take is in favour of its direct dependence on the infective poison; but, in view of the general fact of the intolerance of the peripheral nerves to poisons of all sorts, it is fair to suspect that the post-infectious neuritis may sometimes be due to toxic substances developed from impaired metabolism and sometimes to anæmia and œdema (Class *b*).

It appears to be occasionally possible to induce neuritis by experimental inoculation with diphtheritic cultures, and the form and sequence under which the diphtheritic paralyses clinically occur suggest the action of a specific poison. If, however, the forms under which neuritis typically occurs are considered, it becomes obvious that natural proclivities of the nervous system are at least contributive causes.

The variety of conditions under which the spinal scleroses—both disseminated and system scleroses—occur, suggests predisposing tendencies of one or another kind, common to all and acting in addition to the various special causes. The recent observations of Obersteiner (95) and Redlich, that the narrowing of the posterior nerve-roots at their point of penetration through the pia mater suggests a mode by which slight pressure (as from syphilitic meningitis) could set up the ascending degeneration characteristic of tabes, do not afford a complete explanation of that disease. This theory leaves unnoticed the close analogy between the syphilitic tabes of the lateral columns and that of the posterior columns. The occurrence of the peripheral neuritis met with in connection with syphilitic tabes, but rarely seen at other stages of syphilis, points to the presence of some poison as yet undiscovered, and strengthens the probability that the posterior column degeneration of anæmia and ergot-poisoning, as well as that of syphilis, is mainly of toxic origin.

The complex nature of the infective poisons is shown by the varying relation between the toxic symptoms of the first stage (fever, delirium, pain, &c.) and those of the later stages.

In influenza the fever may be of trifling amount and duration, and yet the post-infectious diseases severe and lasting; in cerebrospinal meningitis the fever is often slight. Again, it has been found possible, experimentally, to neutralise to a certain extent the fever-producing element in bacterial poisons by special anti-toxines. (V. Centanni and Bruschettini, *Deutsch. med. Wochenschr.*, 1894, No. 12, p. 270.)

Finally, it is probable that a routine examination, by the best modern methods, of the central and peripheral nervous system, after infection, would show changes to be present far oftener than is now supposed. The studies of Golgi in rabies lend support to this view.

As an additional contributive cause of lesions, fatigue may be mentioned, since the experiments of Hodge (50) show that this is capable of exciting manifest changes in the nerve cells. It is a question for further investigation whether or not fatigue acts more forcibly on nerve centres poisoned by the products of infectious processes.

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(i.) **Sur les Paramnesies.**—At Lalande, *Revue Philosophique*, November, 1893.

(ii.) **La reconnaissance des phenomènes nouveaux.**—Bourelon. *Ib.*, December, 1893.

(iii.) **Observations sur la fausse mémoire.**—Dugas. *Ib.*, January, 1894.

(iv.) **A propos de la paramnesie.**—J. le Lorain. *Ib.*, February, 1894.

(v.) **Note sur le paramnesie.**—Lapie. *Ib.*, March, 1894.

(vi.) **L'impression de "l'entièrement nouveau" et celle du "déjà vu."**—Duejas. *Ib.*, July, 1824.

(vii.)—**La paramnesie ou fausee mémoire.**—J. van Biereliet. *Ib.*, July, 1894.

(viii.)—**Sulla paramnesia, o falsa memoria.**—T. Vignoli. (Cited by Soury.) *Ib. Ib.*

Little attention had been paid to the subject of paramnesia since the studies of Kräpelin and Burnham until the last year, during which a series of articles have appeared in the *Revue Philosophique* and elsewhere, which, if they afford no definite solution of the problem, have at least recalled attention to its salient features.

To recapitulate, Kräpelin[1] adopted Sully's[2] classification of illusions and hallucinations of memory, parallel to illusions and hallucinations of sense-perception, confining himself to the study of the latter, *i.e.*, total falsification of reminiscence. This he subdivided into (*a*) simple or direct; (*b*) associative; (*c*) identificatory delusions of memory. Paramnesia, in its modern sense, is confined to the third class only. The essentials of true paramnesia are, absolute and total recognition of the scene or situation ("ce n'est pas une autre perception, c'est identifiquement la même"), a painful emotional state varying from mere *ennui* at always living through familiar sensations to acute terror and suffering, and, in some cases, illusory prevision of the future. It is essentially a phenomenon of normal life. Pathological cases of paramnesia are only extreme forms of what occurs under normal conditions, and "the further study of paramnesia will probably furnish admirable illustrations of the way normal mental activity passes over into abnormal." (Burnham, *Am. Jour. of Psychol.* ii., 1889.) It occurs in the waking state, in perfect sanity, full possession of the critical faculities, and yet makes good its distinctive character. Strictly speaking about 30 per cent. are subject to it. It is unaffected by sex and education, is most frequent among the young, or in persons with a lively imagination, is evoked by fatigue or by unusual excitement, and we may note that the phenomenon occurs, *e.g.*, in crowds, or at theatres, under mass-influence.[3]

The theories advanced in explanation of these facts do not impress one as conclusive. The earliest that we have in paramnesia, a real memory of a former existence, may be rejected. Nor does Maudsley and Wiegan's double functioning of the cerebral hemispheres carry conviction. Anjel had a pretty theory of delay between sensation and perception, the mind is affected by sensations before it co-ordinates them; but this would not necessarily produce paramnesia. Lalande (i.) falls back on the double representation of the same image—a momentary interruption and resumption of perception, seeming in the rapidity of thought to be prolonged or projected indefinitely, and further appeals to the "telepathie" sense in support of his opinion. We cannot deny, he says, that there exists an abnormal and unconscious perception, a telepathic sense, due either to the intensification of known senses, or to the rudiment of another, as far beyond them as sight is beyond touch. The unfamiliar excitation of this sense makes us perceive twice over. It can similarly be aroused by dreams, passion, the hypnotic state, and other still unknown physiological agents. Whatever be the surmise adopted, the key to paramnesia lies in the existence of a double perception, at first unconscious, and subsequently conscious.

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[1] "Ueber Erinnerungs Fälschungen, Krapelin." *Archiv. für Psychiatrie.*

[2] "Illusions." J. Sully (International Scientific Series).

[3] Cf. Farde. "Psychologie des Sectes et des Foules." (*Revue des Deux Mondes*, 1894.)

To this Dugas (iii.) replies that paramnesia is only complicated by the introduction of telepathy, and suggests that it is a special case of double personality and perceived at the moment of occurrence. He finds a cause in spontaneous auto-hypnosis, and, unlike Lalande, believes paramnesia to be a neurosis.

Le Lorain (iv.) follows with a short critical note which endeavours to reduce the phenomena to the category of associative memory, but he fails to account for cases of "true" paramnesia.

Meantime Vignoli (viii.) was applying these several theories to cases of paramnesia under his immediate observation. He adduces several striking personal experiences, and brings them under the laws of normal psychical processes, telepathy and hypnotism being superfluous. He proposes three causes: (i.) the awakening of mental images, ideas, and feelings by association; (ii.) the rapidity of thought; (iii.) the constructive power inherent in mind and imagination, and insists strongly upon the latter.

In his second note (vi.) Dugas remarks that the sensations which gave rise to paramnesia are usually characterised by banality, producing an undefined feeling, a sense of familiarity, which Fouillée (introduction to *La Venèse de l'idée de temps*, Euyan) had compared to colour sensations. May there not be two classes—*sensitifs* and *intellectuels*—the former having a vague sentiment of time in memory, the latter its localised representation? It is worth noting, adds Dugas, that paramnesia seems to be allied to coloured hearing, and the faculty of self-abstraction from time and space, as though connoting a general "sensitivity" of temperament.

The last two notes (ii. and vii.) concur in showing the influence of attention on paramnesia. Bourdon proposes to examine the clear state of mind which accompanies cognition, instead of the more or less obscure conditions of recognition. He finds that in any series, the perception of known objects is keener than of unknown, *i.e.*, it is usually the former which are perceived vividly. Hence, when the unknown, or "new" sensation surges up with unusual vigour, we assume that it is known; we tend to "recognise" what provokes attention and interests us. Hence a close relation between attention, perception (or cognition), and recognition, productive of confusion.

Van Biereliet also assumes that the ideas and images which suggest the illusion of paramnesia must be unusually vivid—the subjects being in an absorbed state in which the usual landmarks afforded by the memory of motor images are in abeyance. In this state of "passive" attention new images may arise as freely as represented images—the field of consciousness is momentarily narrowed. The illusion of familiarity persists until the new "sencept" is complete when, normally, its elements are seen to form part of a *new* representation. Motor images are specially predominant in children, and where they are temporarily in abeyance the psychical mechanism must inevitably be disturbed, and illusions follow.

* * *

[NOTE FLETE.]—It must be remarked in conclusion that the last explanation only applies to banal things, to "common" objects. Any keen sensation, such *e.g.*, as crowd upon one in foreign travel, is at once received *as* new, not as remembered. We consciously apprehend it, and extend our mental horizon with no *blasé* sensation of previous experience. Does not the point lie in individual keenness of perception, or, on the other hand, as Vignoli insisted, in the power of anticipation, of imaginative construction, which from time to time coincides with the thing anticipated?

And in this aspect we may admit the imaginative or constructive element in dreams, as quite distinct from the dream illusions. Lalande excludes (*v.s.* Burnham) on the ground that the field of consciousness is so narrowed in dreams, as in insanity, that none of the usual landmarks of association by which we orientate ourselves can be present.

Modern psychology is coming more and more to recognise the existence of different types, or modes, of perception as of memory. (For the last, cf. *Les Grenels Calculateurs.* Binet, 1894.) And as our synthesis of temperament becomes more complete, the phenomena of paramnesia may prove as purely "constitutional" as chilblains or coloured hearing.

ARTICLES ON MEMORY.

Bourdon on Influence de l'age sur le mémoire immédiate. *Revue Philosophique*, August, 1894, gives statistics to show that "immediate" memory (which he correlates with attention and grasp of consciousness) increases from the age of 8 to 20, chiefly between 8 and 14, less from 14 to 20. He investigated 100 children, using series of 6, 7 and 8 figures or letters, and 5 and 6 words, and concludes that as a rule memory is in direct ratio with intelligence.

Ribot on Recherches sur le mémoire affective. *Ib.*, October, 1894, indicates a still unexplored region of memory, in which lie the images of olfaction, gustation, internal sensation, pleasure, pain and emotion in general. Can there be "spontaneous" revival of these images, or does their reproduction depend upon internal stimuli of the same character—an excited revival? Out of 60 experimental cases, 12 gave affirmative answers to spontaneous revival of all images of taste and smell, 48 could revive certain sensations only. Hunger, thirst, fatigue, pleasure and pain, were answered affirmatively. Emotions also, but in most cases this is true of the *intellectual* memory only; the true emotional state of feeling as well as circumstances is much rarer.

M. Ribot concludes that there is an "affective" type parallel to the "visual," "auditive," and motor types, consisting in the facile, complete, and preponderating revival of "affective" representations.

Individual differences of character are largely owing to it, while it proceeds rather from cerebral conditions ("*known or unknown*") than from intensity of primitive impression. To feel an emotion keenly, and to remember it keenly, are two distinct processes. The impression must be deep as well as vivid. Notably there is often an aggravation, or rolling up, a latent inculcation of painful memories, which relates to individual temperament. The article is merely thrown out to supplement the more adequate studies of "intellectual" memory already familiar to the psychologist.

Dugas on La Mémoire brute et la mémoire organisée. *Ib.*, November, 1894, points out that there are two essential types or "species" of memory analogous to the distinction between sensation and perception; besides the "varieties" visual, auditive, &c., more commonly insisted on. These two types he classifies as "rule" (*mémoire brute*), and organised (mémoire organisée). The first is merely the passive repetition of a sensation, involuntary unconscious. The second involves interpretation and selection, conscious mental activity. Memory without effort is not co-ordinated; the mental process is one of ingestion, not digestion. The time sense is an important element in organised memory. The brain is master and not servant of its images. Reason and imagination enter into "true" memory; it is less a sum than a synthesis of images. And in diseases of memory dissolution of form is more fatal than "material" loss; it is not the number, but the nature and kind of facts obliterated that constitutes the gravity of amnesia.

SCHIFF'S "MEMOIRS."

Moritz Schiff on Gesammelte Beiträge zur Physiologie. (*Lausanne Benela*, 1894, Vols. i. and ii.)

Students of neuro-physiology will welcome this collection of Schiff's "Memoirs," of which the second volume has just been published under the editorship of Prof. Herzen. Besides a *précis* of his work on respiration and a number of early papers on vascular nerves, it contains several of his most important contributions to neuro-physiology. It is not a little remarkable to find that many of the vexed questions of the present day were attacked and answered by Schiff twenty or thirty years ago. Buried in some periodical publication they have been forgotten, and are only now rescued and reissued in a consecutive series to witness to the indomitable courage and perseverance of the veteran physiologist. Thirty years ago, before Darwin and others so familiar to us had written on the comparative physiology of plants and animals, Schiff (in an introductory lecture at Florence, 1863) proclaimed reflex action to be the distinguishing characteristic of animal life, evident even when the nervous centre producing it escapes observation. Another article on Cœnesthesia, published in 1871, is reprinted as a contribution to current theories of "consciousness," with special reference to the conclusions of Ribot (*Revue Philosophique*, November, 1892). Many of the papers are critical, and deal with the works of Du Bois-Reymond and Waller. Another series (1877-78) treats of inhibitory nerves. Schiff finds that stimulation of the nerve trunk produces inhibition of its peripheral branches in regard to certain stimulants—*e.g.*, the blood, in the case of cardiac inhibition. This effect he terms *negative irritation*, and describes as "a particular kind of activity of the motor nerve suppressing a muscular contraction which would have been manifested if the nerve had not been irritated" (629). All Schiff's conclusions are supported by experiments, in regard to which he claims a value for the facts discovered, even where the theories leading to them may have to be rejected. (650.)

Vol. ii. treats of the physiology of muscle, cardiac innervation, and innervation of the lymph hearts.

A paper, dated 1858, on "The Direct Stimulation of Muscle," is republished with an appendix of 100 pp., dated 1894, which sums up the "idie-muscular," *v.s.*, "neuro-muscular" controversy, discusses the question whether the constant current, or its interruptions only, is a stimulus, and gives some excellent tracings.

The papers in the second section range from 1849 to 1882. In the third from 1850 to 1885.

As Schiff points out in the preface, the admirable arrangement and editing of these two volumes are due to M. Herzen. It was no easy task to bring such wealth of material, text and tracings into the consecutive form in which they have now been published, and the "Memoirs" will stand as a valuable and permanent contribution to physiology.

FRANCES A. WELBY.

Sensory Nerve of Muscles. (*Landauer.*)—In relation to the somewhat problematical "sense" commonly referred to as the muscular sense, proof of the existence or non-existence of sensory nerves in muscles is still extremely desirable, for the question cannot be considered as answered either by the much-quoted observations of Sachs, nor even by the more recent experiments of Chauveau. Nor, indeed, can they be considered as satisfyingly answered in this, the most recent investigation of the point. Landauer (who, by the way, commits two historical inaccuracies at the outset of his paper—the first attributing to Wundt the opinion that the muscular sense is of central origin, and the second attributing to Bell the discovery that is generally associated with his name) examined frog's muscle by various methods, and says that, besides broad motor nerve-fibres, he found much finer nerve-fibres to all appearance sensory and vasomotor. He comes to the conclusion from finding that subsequent to section of the anterior roots the motor nerves undergo degeneration while the sensory do not. He seems to think that ordinary motor fibres give off finer branches that are vaso-motor. He gives figures of his presumably sensory nerve-endings, but does not really give much proof that muscle, as distinguished from the connective tissue of muscle, possesses sensory nerve-ending.

A. D. W.

Langendorff (*Pflüger's Arch.*, 1894, p. 521) finds that the surprisingly rapid loss of irido-constrictor power of the motor oculi *post-mortem* is due to the intervening ciliary ganglion. Excitation of the third nerve above the ganglion quickly ceases to produce any effect. Excitation of the third nerve below the ganglion has a powerful effect long after death, *viâ* the short ciliary nerves. Constriction of the pupil *post-mortem* is conveniently demonstrated on the rabbit's eye by laying the optic stump across the electrodes; this excites the long ciliary (dilatators) as well as the short ciliary (constrictors), but constriction predominates. The ciliary ganglion, then, is a ganglion traversed by *motor* fibres, and not an analogue of posterior root ganglia.

A. D. W.

Betheiligung der Nerven an der Schwankungen in der Pupillen Wiete. *Pflüger's Archiv.* June 1, 1894.)

Excitation of the cervical sympathetic causes, in addition to the well-known dilatation of the pupil on the same side, reflex constriction of the pupil on the opposite side, presumably due to retinal excitation of the eye whose pupil is dilated. Excitation of the central end of the vagus gives reflex constriction of the pupil on the same side, reflex dilatation on the opposite side. These effects vary with the state of excitation of the animal (cat, rabbit).

A. D. W.

Wundt on Psycho-Physical Interactions.

In a recent number of *Philosophische Studien* (x. 1, *Ueber psychische Causalität, und das Princip des psychophysischen Parallelismus*), Wundt points out that psycho-physical interactions are two-fold: physico-psychical (centripetal) when the external stimulus produces a psychical result in the organism, resulting in *sensation;* psycho-physical (centrifugal), when the psychical process, *e.g.*, will, motives, has a physical effect and produces *movement.* There is thus an empirical basis for psycho-physics, but it is needful to protest against the current tendency to conclude that this covers the whole field of psychology. For example, the parallelism can only apply to processes of consciousness. The physical concomitants of *unconscious* psychical processes can only belong to metaphysics, and again no association of physical processes can explain the nature of the connection between psychical elements any more than our ideas are able to throw light on the physiological processes which accompany them. Wundt criticises sharply the "materialistic psychology" of Münsterberg and Ziehen. These psychologists, he says, throw "matter" out of the window as unthinkable, and bring it in again under the name of "material processes." They affirm matter to be a hypothesis, and yet use it to explain psychological phenomena. The old materialism at least believed in "matter" *per se* and in its molecular motions. Here we have really a new metaphysic, the more dangerous in that it is sheltered behind the theorems of natural science. Experimental psychology, moreover, reduces psychological processes to anatomical elements, until it seems almost superfluous to maintain with Münsterberg that psychology *is* a science. It rather appears as an appendix to muscular contraction. The real subject of psychology is not the physical but the psycho-physical individual. "Bad analogies," concludes Wundt, "are intellectual opiates," and the soul is not explained, though it may be interpreted, by reflex determinism. The rest of the number is occupied by articles on *das Massprincip der psycho-physik,* by F. Lange; *die Abhangigkeit-zwischen Reiz u. Empfindung,* Merkel, part iv., and Dr. Van Biervliet's account of experiments to show that the duration of reaction-times is considerably affected by the pulse-rate.

II. *Die Empfindung als Funktion der Reizœnderung. Ebbinghaus's Zeitschrift für Psychologie u. Physiologie der Sinnesorgans* (vii., 4). Preyer extends Du Bois-Reymond's law of muscular contraction in response to electrical excitation, to all sensory and motor nerves, in response to any adequate stimulus. He finds that the threshold of sensation may be considerably raised with a continuous and steadily increasing stimulus, whereas sudden changes or interrupted stimuli produce sensations at a lower level. The sensation or the contraction corresponds, not to the actual magnitude of the stimulus, but to its alteration from moment to moment, and is also determined by the rapidity and time-duration of the stimuli. The nervous system has in its evolution learned to respond to marked and rapid changes of excitation, rather than to small or steady stimuli. In a former number of the *Zeitschrift* (vi. 472) Scripture had given applications of the same law to changes of sound and of temperature. A very gradual change of stimulus may raise the pitch as much as a whole tone imperceptibly, whereas more rapid changes produce an immediate change of sensation. In temperature changes, also, a gradual rise of 10° C. or more may be unnoticed, although a sudden change of ¼° is perceptible. The unnoticed pressure of the atmosphere is another instance. In *Zwei Fälle von Grunsehen* (*ib.*, p. 305), Somya draws attention to the rare phenomenon of chloropia or green vision. He attributes it to minute changes in the choroid, and not, as has been supposed, to retinal modification.

In regard to the question of central, *versus* peripheral, fusion of sense-impressions in the case of sound, Wundt (*Phil. Stud.*, viii., 641, 1893, *Ist der Hor-Nerv direct durch Tonschwebungen erregbar*) sums up the arguments of Scripture and Karl Schäfer, as well as the earlier work of Cross and Goodwin, and Sylvanus Thompson. The conclusions of Scripture (and also of Ewald) run counter to the theory of specific nervous energies, according to which each auditory fibre can convey one tone only, whatever the nature of the stimulus. Wundt refers to other analogous phenomena equally unfavourable to this theory, *e.g.*, the beats and beat-tones of König, which indicate that beats can arise in the brain without interference at the periphery, and that direct periodical stimulation of the auditory nerve can, at a certain frequency, give rise to tone-sensations. Even if we accept the theory of Helmholtz, it connotes a mechanical excitation of the nerve-fibres at the organ of Corti; and it is equally conceivable that direct excitation of the nerve might occur without peripheral intervention. Ewald, in his investigations on the end organs of the auditory nerve, found that pigeons whose labyrinths had been extirpated still reacted to auditory stimuli, whence he concluded that the sound waves directly stimulated the stump of the nerve by transmission through the bones of the cranium. A later series of observations (*Die centrale Entstehung von Schwebungen zweier monotisch gehörten Töne*, *Pflüger's Archiv.*, June 1, 1894) seem to justify the same conclusion in regard to man. It would follow that when deaf people hear sounds conducted through the cranial bones this is not a proof, as is generally supposed, of the integrity of the labyrinth. If the trunk of the nerve is capable of direct stimulation, the labyrinth might in these cases be completely disorganised. Again, if the nerve be thus directly excitable, the case for a central origin of beats is greatly strengthened, seeing that interference in the terminal apparatus is not an indispensable condition of their production.

K. Schäfer maintains that the bones of the cranium conduct sound from one ear to the other, thus making peripheral fusion. Ewald, however, arranged two tuning-forks so that they could each be heard by one ear only. One was tuned to give 130 vibrations per second, the other to a slightly higher rate. Heard separately there was no indication of oscillation; heard together there was a distinct and easily-counted beat. The conclusion is that sounds conveyed separately to each ear may be *centrally* fused into a beat. The beat moreover coincided with subsequent binaural observations with the same tuning forks. The case seems to stand upon the same footing as the analogous case of the eye, in which the balance of evidence is to the effect that two complementary colours presented separately to each eye may give rise to the sensation of a single resultant colour, the mechanism of which fusion must obviously be central.

Another interesting analogy between eye and ear is that established by Zwaardemaker (*Das presbyacousische Gesetz Zeitschrift für Ohren-heilkunde.* June, 1893. *Der Umfang des Gehörs in den verschiedenen Lebensjahren. Ebbinghaus Zeitschrift.* March, 1894) who formulates a law of what he terms presbyacousia. He gives a curve founded on 219 observations to show that the range of hearing of the upper notes diminishes from *e* 7 (41,440) max. in infants, to *a* 6 (28,160) or even *g* 6 (25,344) in adults. He refers to an earlier paper in proof of the same limitation in regard to the lower notes, which, however, he admits to be far more uncertain. In short, from the age of 4 to 70, hearing, he says, undergoes a continuous restriction, losing as much as half an octave. In the second paper he alludes to the inductively established fact that bone conductivity decreases with age. He connects the beginning of the decline curve of hearing with the end of bone-growth.

FRANCES A. WELBY.

Association by Unconscious Middle Factors. Jerusalem, Wundt.—*Phil. Stud.*, x., ii.

Dr. Jerusalem communicates an experience of Herr Baumgarten, purporting to show that the idea or memory-image of two people, vividly called up by the unnoticed scent of a flower (which was not detected till after the memory-image had arisen in consciousness) implies that "the smell-sensation was here a really unconscious middle factor." He adduces this in support of Scripture's views (*Phil. Stud.*, vii.).

Wundt rejoins that the scent of the flower was "unrecognised" (*unbemerkt*), not unconscious (*unbewusst*). Baumgarten's observation is interesting, because the unrecognised middle factor is here present in consciousness, whereas in Scripture's experiments the middle factors were not direct sensations during the process of mediate association, but were reproduced (vii., p. 78). Here the scent was *perceived*, though not *apperceived*, *i.e.*, not distinguished from the scent of the other flowers. Wundt, therefore, calls this "a case of mediate association through middle factors present in consciousness, but only dimly perceived there."

FRANCES A. WELBY.

Dana and Ketscher on Paralysis Agitans.

I.—In some preliminary remarks on the clinical phenomena of the disease, Dana (*New York Medical Journal*, June 10, 1893), emphasizes three classes of symptoms.

(1) Motor symptoms, chiefly due to difficulties in commencement of movements, or, as the author terms it, "a hitch in volition."

(2) Vasomotor symptoms, flushings, sweats, subjective sensation of heat, peculiarities of pulse. He believes there is a general vasomotor paralysis, affecting the vessels of the cord and the nerve trunks, in addition to those of the skin.

(3) Disturbances of the blood, and of metabolism. Purpura hæmorrhagica occurs sometimes, glycosuria sometimes; and in the opinion of some authors, phosphaturia. The author examined the blood in two cases; in one, the chief abnormality was absence of eosino-philous cells; in the other, there was "anæmia of chlorotic type; lessened percentage of hæmoglobin, and decreased resistance of red cells."

(4) Sensory symptoms, viz., sciatica, neuralgia, and rheumatic pains; distressing sensations of heat and cold.

He then turns to the pathology of the disease with which his paper is chiefly occupied. He quotes 44 cases of paralysis agitans, real or supposed, in which *post-mortems* have been made, but out of these he rejects all but fourteen, as being in his opinion scientifically valueless. Upon the cases of Dubief, von Sass, Koller, and Borgherini, he places most stress. In the bulk of these cases certain anatomical facts appear, which although rather vague may possibly be taken as the basis of the morbid anatomy of this disease. They are degeneration of the vessels, enlargement of the perivascular spaces, proliferation of the neuroglia and connective tissue in the neighbourhood of the vessels; pigmentation and some degeneration of the spinal motor cells, and of those in the bulbar nuclei; thickening of the pia mater surrounding the cord, with sclerosis of the neighbouring periphery of the cord; blocking of the central spinal canal by proliferation of cells; changes in the cells of the sympathetic.

The author's own case is as follows:—A woman, age about 58 at time of death, died from exhaustion five years after onset of the disease. The symptoms began after a fright, and consisted of pain and weakness in the back; tremors of the limbs, followed by weakness; stooping gait; slowness of movement, &c. The brain and cord were hardened in Muller's fluid. In the cortex cerebri was some increased vascularity, dilatation of the vascular spaces; and some trivial changes in the nerve cells.

In the nuclei of the seventh, ninth, tenth, and eleventh cranial nerves was pigmentation, with some degeneration of the cells.

In the cord, the pia mater, particularly of the cervical and lumbar regions, was thickened; while in the lateral columns beneath was some diffuse infiltration of connective tissue, which had not however caused much degeneration of the nerve fibres. The cerebellar tracts were not affected by this sclerosis. The vessels in the anterior cornua were much dilated, particularly the veins. The anterior cornual cells were somewhat shrunken and pigmented, and took stains irregularly. The central canal, at some levels, was filled with *débris* and exsudate. The anterior nerve roots were congested, and perforated with dilated vessels.

In a second case, the details of which are not fully given, the author found marked thickening of the spinal pia mater; with diffuse interstitial sclerosis, of the lateral columns especially; congestion of the central and anterior gray matter; and slight changes in the nerve cells.

The anatomical seat of the disease the author places in the spinal cord, medulla and pons, and more particularly in the blood vessels of the central and anterior gray matter of the cord, and in the lateral columns (pyramidal tracts, lateral fundamental columns, and lateral limiting layers). As to the nature of the anatomical change we quote his own words :—"This anatomical basis I believe to be essentially as follows: First, a congestion of the central and anterior parts of the gray matter of the cord with some thickening of the blood vessels, but not very much, and some changes of the external wall of the nature of periarteritis. With this congestion and vascular irritation there occurs a proliferation of connective tissue, which especially invades the lateral columns of the spinal cord. In severe cases it affects also the periphery, producing a leptomeningitis and a zone of sclerosis surrounding the cord in a variable degree. The congestion with dilatation of blood vessels affects especially also the anterior roots, to a less extent the posterior roots. There is degeneration of the cells of the anterior horns, which, however, occurs only late in the disease and does not affect all the cell groups. The central cells or the cells of the median and central areas, which we suppose to be more connected with vasomotor and glandular functions, seem to be especially involved. There is also—and this is an important fact to bear in mind—a decided loss of the fibre net-work in the anterior horns and the central gray matter. The changes appear to involve especially the cervical and lumbar regions. Similar changes occur in the medulla and pons, but here they are less marked. Degenerative changes, however, are to be found, in many cases at least, in the cells of the nuclei of the glosso-pharyngeal and pneumo-gastric nerves. Such changes would explain the disturbances in the vasomotor function and heart action found in the disease."

He makes this further suggestion as to the causation of the disease, viz., that it is due to the presence, in the circulation, of some irritative toxine, which in its turn may originate in some glandular defect—the process being analogous to that of myxœdema. For this hypothesis, however, no evidence is adduced.

II.—Ketscher's paper (*Zeitschrift für Heilkunde*, 1892, p. 445) opens with a very complete account of the work already done upon the morbid anatomy of paralysis agitans. It would appear that, after dismissing those cases which probably were not genuine paralysis agitans at all, the remainder consist partly of cases in which no morbid appearances were discovered *post-*

mortem (some of which, however, were not examined microscopically), and partly of others in which more or less definite results were obtained. These results may be summarised as—hyperplasia of the connective tissue and of the neuroglia in the spinal cord; changes of various degrees in the vessels, pigmentation of the ganglion cells, blocking of the central canal of the spinal cord, presence of corpora amylacea in more or less excess. Similar alterations have also been found in the brain, the peripheral nerves and muscles, and in the sympathetic system. Most investigators consider that the chief seat of the disease is in the spinal cord; though they are by no means unanimous on this point; and some distinguished men (Charcot, Oppenheim, &c.) maintain that paralysis agitans is a "pure neurosis."

Ketscher investigated microscopically three cases of paralysis agitans; the ages of the patients were respectively 70, 68 and 76. In all three cases he found changes in the central nervous system (mainly, it would appear, in the cord); in two cases changes in the muscles, in one case in the peripheral nerves. These changes he summarized as follows:—

(1) As to the parenchyma.

Scattered indication of degeneration and atrophy, viz., in the ganglion cells of brain and cord—pigmentation, indistinctness of outline, absence of nuclei, finely granular degeneration; in the nerve fibres (particularly of the posterior columns of the cord)—changes ranging from mere swelling of the axis-cylinder to complete disappearance of the nerve fibres; in the muscles—multiplication of nuclei, loss of transverse striation, hyaline and fatty degeneration, disappearance of muscle-substance.

(2) As to the interstitial tissues.

There was general increase of the interstitial connective tissue. Around the cord the peripheral connective tissue was thickened, and broad bands extended from it towards the central parts, particularly in the posterior columns. In the peripheral nerves, the peri-neurium was thickened, and in some places the endo-neurium also. Similarly the connective tissue between the muscular fibres was thickened.

The neuroglia both of brain and cord was increased. The neuroglia layer of the cerebral cortex, and the ependyma of the ventricles was thickened. So was the glia of the cord, both the peripheral layer and that of the internal parts, and this overgrowth appeared to start from the neighbourhood of the vessels. In the posterior columns particularly this overgrowth penetrated not only between groups of fibres, which were thus marked off into separate bundles, but also between the individual nerve fibres in some places. The disease reached its maximum in the lumbar region of the cord. There were numerous corpora amylacea, in the ependyma of the lateral ventricles, in the superficial parts of the brain, in the peripheral parts of the cord, and along the posterior fissure of the cord.

The central canal of the cord was blocked with proliferated epithelial cells.

(3) As to the vascular system.

The vascular walls were thickened. Here and there were miliary aneurysms, or even small extravasations of blood. The adventitia of the cerebral vessels contained a good deal of pigment. The lymphatic spaces—peri-vascular and peri-cellular—were dilated, and contained sometimes coagulated lymph, sometimes leucocytes, sometimes blood. Here and there in the neighbourhood of these spaces an "œdematous softening" of the brain substance could be made out.

These changes, which have been observed by others besides himself, the author believes to be no fortuitous occurrence, but to stand in some causal relation to the disease.

A certain reservation has, however, to be made in view of the author's investigations into the condition of the nervous system in aged people generally. He made microscopical examination of the nervous system in ten aged people, dying from various causes; and comes to the conclusion that in old age changes are found resembling in kind those found in paralysis agitans, and differing only in their less advanced degree. He finally expresses his agreement with the view of Jacobsohn, Dubief and Borgherini, that paralysis agitans is nothing else than senility of the nervous system, early in occurrence and intense in degree.

J. A. Ormerod, M.D.

BRAIN.

PART IV., 1895.

Original Articles and Clinical Cases.

STUDIES ON THE LESIONS PRODUCED BY THE ACTION OF CERTAIN POISONS ON THE CORTICAL NERVE CELL.—I. ALCOHOL.

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PART I.

Section I.—Experimental Lesions produced by the Action of Absolute Ethyl Alcohol on the Nerve Cells of the Rabbit's Brain.

THE most extensive treatise on the pathology of chronic alcoholism to be found in the English language, is Bevan Lewis' article in his text-book on "Mental Diseases." Indeed, so widely has it been accepted as the minute anatomy of this morbid state, that in Tuke's "Dictionary of Psychological Medicine," it is quoted to the exclusion of all other writings. Abstracted, it reads as follows:—"The changes in the vessels are nuclear proliferation, atheroma, and aneurysmal dilatation, the last eventually giving rise to a cribriform condition. The motor cells are swollen and rounded, stain deeply, become granularly pigmented, and the apical processes degenerate. The cellular wall is thickened. There is a considerable quantity of pigment deposited between the shrinking protoplasm and this cell wall. The processes of the cells are stunted, and are covered with nuclei, and the protoplasm is granular or vacuolated. In the lowest layer the scavenger cells and nuclei cover the spindle nerve cells, which are very much degenerated and altered, and are practically being devoured by these proliferating cells.

"The medullary sheath of the nerve processes gradually disappears, or is so altered by the invading connective tissue that the axis-cylinder—which is frequently fusiform, as in other cases of inflammation of the nerve fibre—can be perfectly well stained with the aniline colour, when it becomes a prominent feature in the cortex."

Lewis describes the motor cells of the so-called third layer as being the first implicated in the cortex, and that those of the deeper and more superficial layers follow in order.

It would seem from this description as if there were no specific lesions of the central nervous system, from the continued abuse of alcohol, to be discovered; certainly, the vascular changes found are frequent in brains that have exhibited no clinical picture of the effects of alcohol during life, and taking atheroma and miliary aneurisms from among the list, it would appear as if there were other conditions of which we at present know nothing; as all pathologists are familiar with brains that show in marked degree these changes, but whose owners have in no wise been addicted to the abuse of the drug. Nevertheless, there can be no doubt that alcohol, in the common forms of wines and distilled liquors, does exert a direct influence on the cerebral vessels, irritating their coats, especially the intima: perhaps paralysing the vaso-motor nerves accompanying them in more or less enduring fashion, hence giving rise, from the irritation, to nuclear proliferation, and from the distension of the vessels, to transudations, local minute extravasations, clumps of hæmatoidin granules, and detritus in the perivascular sheaths; then blocking of the lymph currents, and general disturbance of the lymph flow in the peri-vascular and peri-cellular channels.

Of the changes in the nerve cell produced by the direct contact of the diluted drug flowing with the nutrient serum from the blood channels into the peri-cellular spaces, we at present know nothing; but from a clinical standpoint, the immediate effect in occasioning disturbed metabolism must be very great.

With Lewis' ideas of the coarser changes produced by the continued abuse of the drug upon the nerve cell, we are in unison, as only too frequently in alcoholic brains have we met with the shrunken protoplasm, stunted processes, and granular cell bodies; but these alterations mark only the after-effects of long continued malnutrition and irritation of the cells by the poison. In other words, we see only the later effects of a degeneration which has preceded a considerable distance toward utter deterioration of the cell body before it is visible to carmine, hæmatoxylin, aniline blue-black, and the freezing methods. What we really wish to ascertain is not only the condition of the protoplasmic body, but also the early changes in the finest twigs of the dendrites, and the finest variations of the nucleus and nucleolus from the normal.

A newer though very small literature has sprung up since 1890, when the work of Lewis first appeared, and the methods of histological research have been extended to a realm whose boundaries were only beginning to be entered upon at that date. I refer to the beginning of the extended use of the chromate of silver methods, and the magenta and methylene blue stains of Nissl—the one almost fully supplementing the other, in so far that by one we may obtain perfect impregnations of the neuron to the uttermost limits of the dendrites, and by the other an accurate view of the condition of the cellular protoplasm, and in particular the nucleus with its chromophile particles.

The newer literature, as already mentioned, is very scant; portions of three articles only treat of the lesions of the nerve cell when subjected to the presence of alcohol in the living organism.

Colella (*Arch. Ital. de Biologie*, 1894, p. 216), by the application of the Golgi silver method to the cortex of an alcoholic brain, found lesions of the neuraxon of the nerve cell, slight involvement of the cellular body, and dendrites in the alteration. He speaks of the blood vessels and neuroglia as not being implicated in the changes.

Vas (*Arch. f. Exper. Path. u. Pharmakologie*, Bd. xxxiii., S. 141), in an experimental work on the poisonous effects of alcohol and nicotin on the nerve cells, finds well-marked changes in these bodies, especially in the great cells of the anterior horns of the spinal cord, and in the spinal and sympathetic ganglia. As a first change, the cells lose their chromatin structure; the finely granular character is perceptibly diminished, and gives place to a homogeneous swelling. In advanced cases of the alteration, the cellular protoplasm alters its power of taking up the colouring matter, and appears darker than normal. A shrinkage of the protoplasmic body cannot be definitely ascertained.

The study of Andriezen (BRAIN, 1894) is of greater interest than the foregoing works, and is worthy of careful consideration. Using a modification of the Golgi method, he finds extensive changes in the neurodendrites and neuraxons of the nerve cells of the cortex, the alteration beginning in the fine bead-like "contact granules" upon the substance of the protoplasmic extensions, proceed to moniliform swellings along the course of the protoplasmic twigs. With the further progress of the lesion, these softened and enlarged protoplasmic masses form irregular botryoidal masses, mainly clothing the now irregularly bared protoplasmic stem, which rapidly becomes attenuated.

"The changes in the cell body are of similar nature, and consist in a gradual disintegration after the apical and others of the processes have suffered. Here and there at the side of the cell body the protoplasm seems to become frayed, or as it were eroded. In other cases the cell protoplasm becomes vacuolated from within, and the process continues until the whole interior protoplasmic structure is channeled by holes and seams of liquefaction."

The nerve fibres of the cortex Andriezen finds granular and wrinkled in outline, and what is much less certain, a process of unequal staining, portions of the filament being completely stained, while others are pale or colourless, and only visible by the highest powers of the microscope.

It will be noticed that in none of these articles is there any mention of the state of the nucleus and nucleolus. This partly arises from the methods of staining used in the various investigations. Lewis' aniline blue-black does not allow the interior structure of the cell to be accurately seen, nor does the silver method permit any inquiry in this direction, and Vas, who used nuclear stains, does not seem to have considered the nucleus of the nerve cell, probably from the difficulties that attend its accurate investigation. The material at our command has, however, allowed of more accurate procedure, and in this direction our labours have been partly directed, somewhat to the exclusion of the intimate structure of the nerve cells protoplasm.

Thanks to the kindness of Prof. Welch and Dr. Friedenwald in furnishing me with the brains of a number of rabbits that had been subjected to the continued—*intra vitam*—action of absolute ethyl alcohol over a considerable space of time, our material allowed us a better opportunity for the investigation of the nerve cells than could be obtained from human alcoholic brains, in so far that we had an accurate knowledge of the amount of alcohol the animal had been subjected to per kilo of bodily weight, the duration of the poisoning, and last, though not least, we were enabled to obtain the cerebra in much fresher condition than could be obtained from the autopsy table. Appended is a condensed table of the histories of the five rabbits used in the investigation.

CONDENSED RABBIT HISTORIES.

No. of Rabbit.	Apparent Age.	Weight in grammes.	Quantity of Alcohol fed per diem.	Began to feed.	Died.	Weight at time of death.	Clinical cause of death.	Gross anatomical lesions found.
6	Adult	1450	5 to 8 cc.	Dec. 19, 1893	Dec. 20, 1894	1420	Convulsions	Fatty heart
10	"	1920	" "	Dec. 30, 1893	Oct. 27, 1894	2500	Ascites	Cirrhosis of liver
39	"	1540	" "	July 5, 1894	Dec. 5, 1894	1400	Convulsions	Fatty heart
41	"	1890	" "	July 5, 1894	Feb. 4, 1895	1420	"	" "
42	"	1940	" "	July 5, 1894	Feb. 10, 1895	1640	"	" "

Methods of Examination.

All the specimens of brain tissues that came into my hands for the purposes of this study—pathological as well as normal—were hardened either in alcohol or in Müller's fluid. For the Nissl method and modifications thereof used for the demonstration of the cellular structures, and for hæmatoxylin-eosin staining for the blood vessels, the former method of hardening was applicable, but for studying the finer dendrites of the nerve cells it became necessary to devise some ready method of staining by which impregnations with metallic salts could be obtained for tingeing the finer extensions of the nerve cells. A research among the literature devoted to microscopic technique failed to afford any suitable process, and after consideration, the following process was adopted and found to be more satisfactory than any other, and, indeed, it gave finer and more complete pictures of the cortical neurons than the ordinary Golgi method. We have at this time stained a large number of pieces of brain substance with it, both human and rabbit, and have yet to record a single failure in the process; though, of course, all the impregnations were not equally good. The sole disadvantage the method has against the Golgi-Cajal procedure is, that the thicker dendrons are rendered so brittle by the silver that they are readily broken into fragments by the sharp edge of the knife passing through the tissue, but as the fragments are not usually carried far from their natural seat, errors from this source are to be avoided with ease.[1] On the other hand, the great advantage that accrues to the pathologist from being able to make use of any desired portion of an entire brain already hardened after the usual manner in Müller's fluid, is at once apparent, particularly when the silver impregnations obtained are much finer than those had after a troublesome and particular hardening process, and though it is but a small consideration, it is more economical of osmic acid than the Cajal method, as the osmic acid mixture may be used more than once.

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[1] We found in later preparations that the cause of this brittleness was overhardening in the Müller's fluid, and could easily be avoided.

The staining obtained is much more intense than is found after the ordinary chrome-silver staining, the cell bodies and processes are intensely black, and it is only at the points of the finest dendrites that the familiar reddish-black colour makes its appearance. It is evident from the appearance of the nerve cells and axons that the chemical combination between the chrome saturated nerve tissues, and the silver salts is quite different from the simple chrome-silver combination of the Cajal process. Our method is a much closer approach to a true stain than an impregnation, and no traces of an incrustation are ever to be found. Under intense illumination the cell body is found to be homogeneously blackened, and fine lines are seen running through the protoplasm from one pole of the cell to the other. The addition of sodium sulphate and phospho-molybdic acid to the hardening and staining fluids respectively, makes the chemical reaction between the constituent elements much more complicated than when there are only the simple kalium bichromate, osmic acid, and silver nitrate to deal with, and an attempt to formulate the resulting reactions would be most difficult.

The process is as follows:—The cerebra are treated with Muller's fluid until the tissue is of sufficient consistency to admit of fairly thin sections. The portions of the brain selected are then cut into pieces not more than three millimetres in thickness, and the slices are immersed in a mixture of a three per cent. solution of bichromate of potassium, and solution of one per cent. osmic acid, in the proportion of one hundred parts of the former to twenty parts of the latter. In this mixture the slices lie from three to five days, are then removed from the fluid, and slightly dried on filter paper to remove any superfluous bichromate, are washed for a few minutes in a weak solution of silver nitrate, and then go into the second or staining mixture, which is made by adding two drops of a ten per cent. solution of phosphomolybdic acid to each sixty cc. of one per cent. arg. nitrate solution in distilled water.

The second solution is to be made only as needed, and at the moment before placing the brain tissue in it. In the mixture of finely precipitated phosphomolybdate of silver held in suspension in the free nitrate of silver solution, the tissues are immersed, and allowed to remain without being disturbed for two or three days, or if it is inconvenient to cut them at this time, a few drops of fresh silver solution may be added to the old fluid, and in this the tissues may remain indefinitely. The object in adding the additional silver nitrate is to prevent any chance of all the salt being precipitated or absorbed, as plain water will soon cause a deterioration of the distinctness of the impregnation of the nerve cells. Light does not seemingly affect the tissues unfavourably.

The individual details of the component parts of the neuron are somewhat finer than by the Cajal method; each element stands out clearly and distinctly, the axons with their collaterals are clear, and the gemmulæ, or buds on the protoplasmic processes, are fully and equally impregnated, and appear in their proper relations to the parent dendrite. There is always some coarse precipitate along the margins of the sections, but rarely the finer one that is so annoying in the older chrome-silver specimens.

The Lesions found in Preparations hardened in Absolute Alcohol.

This first part of the study was conducted on the brains of two rabbits, hardened in absolute alcohol, and stained according to the methods of Nissl, and with hæmatoxylin-eosin.

In order to fully appreciate the structure of the pathological nerve cell, stained by the Nissl procedure, we must first glance at the normal cell as seen in the control preparations. The aniline dye does not stain all portions of the cellular protoplasm equally, but leaves clear spaces between others more deeply stained—the chromophilic particles. In the cell body the arrangement of these chromophilic particles is always definite, though varying considerably according to the character of the cell itself. Thus, in the anterior horns of the spinal cord we find a net-like arrangement of the stained particles (arkyochromic cells), while those of the cerebral cortex have them mainly arranged in rows (stichochromic cells). Furthermore, these various cells are divided into several main groups by the different relations of the nucleus to the cellular protoplasm. Only two of these groups are ordinarily to be seen in the cortex, mainly cytochromic cells, or nerve bodies having a nucleus approximately the size of a leucocyte, and with a very small amount of stained protoplasm surrounding it, and somatochromic cells having a nucleus of moderate size surrounded by a considerable body of protoplasmic substance with a definite contour.

With the cytochromic cells we have but little interest—the main body of the cortical cells being somatochromic, and of the stichochromic variety.

Furthermore, the nuclei present variations in their size, contents, refraction, and receptivity to the absorption of the aniline dyes. The arrangement of the contents of the nucleus also varies considerably. In some we find only a single well-defined nucleolus, the edges smooth and rounded, surrounded by very fine dust-like grains, all deeply stained by the dye, the whole imbedded in a transparent karyoplasma that does not take up any of the aniline. Others have two or three nucleolar particles; seldom more than this number, all smooth, and situated in the several quarters of the nuclear circle, imbedded amidst dust-like grains of a finer or coarser character.

Besides these several varieties, a third exists, in which the nucleolus is less distinct than in the others, and the nuclear dust is coarser than in the other varieties. All these forms present little difference in their staining qualities, the amount of dye taken up outside the nucleoli and dust is very small, and the nucleus, in consequence, is invariably refractile and light coloured. The fourth variety of nucleus is differentiated by the variation in the receptivity of the staining fluid. It absorbs a little more of the dye, sufficient to colour it slightly, but the numbers of these cells in the healthy brain is minimal, and for the practical examination they may be excluded.

Histology of the Absolute Alcohol Sections from the Alcoholic Brain.

The two rabbits of this series have to be considered separately, on account of slight anatomical differences.

Vessels.—The capillaries and intermediary vessels in the control specimens have a smooth, regular contour; the nuclei in the walls are sharply stained, and take up an equal quantity of the colouring matter, and the cell structure between the nuclear points is free from a suspicion of colour (eosin specimens do, however, have a slight staining of the capillary wall with the dye). The perivascular spaces are narrow but distinct. In rabbit No. 10, the smallest arteries have an altogether different appearance from the control sections. The walls of many of them are irregularly shrunken, at intervals almost botryoidal in appearance, and the nuclei of the vascular walls, while not increased in numbers, are swollen, absorb more of the aniline stain, and project considerably into the lumen of the canal. The perivascular space is larger than in the control preparations, and the hyaline membrane separating it from the cerebral substance is a little more definite than normal. As we progress upward in the scale of size, the changes of an abnormal character do not increase greatly, except that the vascular walls are slightly thickened, while the irregularities in their boundaries are gradually lost; but, on the other hand, the nuclei, especially those of the outer layer of the vessel, are somewhat multiplied—at times, though rarely, sufficiently so to cover the exterior of the vessel with rather dense aggregations of round nuclei, through which the proper structure of the canal is seen with difficulty. The peri-vascular spaces around these vessels are larger than usual, and the cerebral edge of the space stains more deeply than normal. Occasionally there are a few grains of hæmatoidin *débris* within the space; more commonly it is vacant. On transverse section, the walls of these vessels appear to be a little thickened—the change affecting the intima more than the media or externa. Very few of the arteries show any very distinct changes. The larger veins do not show any alterations from the normal. Four or five miliary hæmorrhages, into the sheaths of the vessels, were noted in the several sections, all near the pial margin. Altogether, few definite lesions could be determined in the blood vessels—the perivascular dilatations and hæmorrhages excepted.

In rabbit No. 10, the vast majority of the nuclei of the somatochrome cells of the cortex show similar alterations and departures from the standard established by our control preparations. The most prominent of these changes lies in the central nucleolar figure, for in the place of the smoothly appearing dot, in or near the centre of the ring, it now appears roughened, spongy, or even with elongated projections from its surface (compare photos. 1 and 2). Not only is the nucleolus roughened, but it is also considerably enlarged, occupying now from an eighth to a sixth of the interior of the nucleus—its projections extending to the periphery of the nuclear ring. Around these nucleolar figures are grouped a small number of nuclear dust particles resembling an irregular circle, but the remaining portions of the nucleus are comparatively free from the presence of molecular particles, in contrast to the control preparations. In the clear karyoplasma there is a decided tendency to take up more than normal of the aniline stain; so marked does this tendency become at times that the contents of the nucleus are rendered indistinct, and the body becomes much less refractile than natural.

The above description applies exclusively to the mononucleolar variety of nuclei. The varieties showing two or more nuclei have nearly disappeared, and it is highly probable that the largest masses of spongy nucleoli of the altered nuclei are formed by an agglutination of these chromophilic masses, for indeed in not a few of the cells enlarged and swollen nucleolar particles are seen approaching each other in some portion of the nucleus.

Hardly any nuclei are found differing widely from these two pathological varieties, though not an inconsiderable number of the normally disposed nuclei are seen here and there in the field, the proportion, however, being inconsiderable.

To recapitulate, we find alterations of the nuclei chiefly in the disposition, size, and irregularity of contour of the nucleolar chromophilic particles.

Rabbit No. 39.—The blood vessels show the same condition as in rabbit 10, only not so accentuated. The botryoidal appearance is not so marked. The nuclei of the intermediary vessels are only multiplied to a small extent, an alteration that extends to the smaller veins. In one small artery I detected a distinct aneurysmal dilatation.

All the nerve cells take up a larger quantity of aniline dye than normal, the chromophile masses of the protoplasm are not distinct, the nuclei are nearly all mononucleolar, and the nuclear substance stains a deeper hue than in the control. The nucleoli are comparatively seldom swollen or enlarged, here and there one is seen, perhaps an average of one in fifteen cells, but in many others there is a tendency to be out of proportion in size to the surrounding ones. The nuclear molecules are fine, while the substance itself takes up an unusual quantity of the stain.

A few sections of the cortex of this rabbit were stained by the Weigert-hæmatoxylin method, but I could detect no variation from the normal among the fibres of the coarser bundles of nerve filaments. The finer intercellular fibres were not very numerously stained.

The Normal Structure of the Rabbit's Cortex as seen with the Silver Method.

The arrangement of the nerve cells in the cortex of the rabbit's brain differs little from that of other mammalia. The structure consists of three indistinct layers of cells, with beneath them a rather thin mass of white matter. Viewed with a low power there is but one distinct cellular layer. In the outer or molecular lamina are situated a minimal number of small fusiform and angular cells whose axis-cylinder processes seem to course parallel with the surface of the brain. Beneath this region lie bicornuate, small pyramidal, elliptical, and irregular cells, their axons nearly all turning downward. Beneath these cells lie others of approximately pyramidal form, but of a larger size than those immediately superjacent to them, and mingled among them are a few irregular cells. All the neuraxons from this region turn downward into the radial bands of fibres. The apical dendrites of nearly all the large pyramidal cells reach past the middle of the molecular layer, and by division form an intricate meshwork of protoplasmic extensions beneath the pial limit. The dendrites of the irregular cells spread out to a greater extent laterally than vertically, but without definite course.

Beneath the pyramidal cells having long apical processes, and bordering on the white medullated fibre layer, lie cells of quite a number of forms, mainly irregularly, pyramidal and angular. The stronger processes of these cells are upturned, while the axons are down turned. Situated between these cells and far more numerous, are small irregular cells, with protoplasmic extensions outstretched in all directions. Their neuraxons are all turned towards the surface of the brain, and on their way thither they give off numerous collaterals, and finally reach the tangential band beneath the pial surface, among whose fibres they are eventually lost.

All the pyramidal cells of the cortex of whatever kind, and very many of the irregular and angular ones have, coming off from their protoplasmic extensions, short rectangular or oblique projections or *gemmulæ*, that have been so universally found by the silver method in the brains of all animals, as not to admit of the possibility of a doubt that they are present during life and are not simply artifacts as was once supposed (Koelliker), indeed they have been demonstrated in particularly fine Nissl preparations (Lenhossék), and are undoubtedly a factor of considerable importance in the economy of the nerve cell, though their function at the present time is somewhat in doubt. On the apical processes of both the long and short pyramidal cells, the gemmulæ are usually simply roughenings on the process near the basal end, higher up they lengthen out, and have a bead-like ending. On the thinner branches of the cells, especially those that come off from the basal extremity, they are longer, thinner, and are arranged with considerable regularity, and give to these dendrites something of a feathered appearance.

Besides the gemmulæ, the nerve cells have, upon their branches, a small, though very variable number of varicosities. On the thicker portion of the apical processes, these varicosities are never present, but in the finer dendrites they are here and there to be met with, particularly at the branchings of the stems. These varicosities must be held to be normal, as they are to be demonstrated in methylene blue as well as in silver preparations.

With the Purkinje cells of the cerebellum, the gemmulæ contribute greatly to the actual outspread of the dendrons. They are arranged with great regularity, and endow the extensions with more of a feathered appearance than those of the cerebrum, but differ from those belonging to the pyramidal cells anatomically, in that they have few of the beaded endings peculiar to these psychical cells. Knots upon the extensions of the stems of the Purkinje cells are frequent, are of small size, and give the branches an uneven appearance.

Along the outer edge of the cortex numerous forms of support—neuroglia cells, such as have been described by Retzius and others, are seen, but lower, among the nerve cells, the glia elements are but sparingly stained. Few of them belong to the long-rayed Golgi type, but are replaced by a cell, with quite large body, and short but thick, and knotty arms. These cells are always to be seen in the immediate vicinity of an intermediary blood vessel, which they frequently surround, and send off to its hyaline sheath one or more thick processes that terminate in knob-like endings. In disease, unlike some other varieties of neuroglia, they atrophy and disappear.

The Pathological Histology of the Rabbit's Cortex in Chronic Alcoholism.

The material for this portion of the study comprised the brains of three rabbits preserved in Müller's fluid. The lesions found in all the specimens were identical.

In formulating any description of the pathological changes found in chronic alcoholism, we should always have prominently before our mental vision, the fact that the normal characteristics of the cortical cells as we see them by any of the more usual methods of preparation, vary within certain fixed limits, and that these must be overstepped before we can say with certainty that we have before us an abnormal condition. By any method of staining with the chrome-silver salts this is perhaps more strikingly the case than by other methods, and the observer must be constantly upon his guard against deceptions into which he may very innocently fall.

A portion of the uncertainty of the silver method we have been able to obviate from the fact that the new mode of staining we have pursued is more fixed and reliable for the adult brain than any other treatment we have hitherto tried; truly a comparison of many scores of normal cells in the control slides show little or no variations in the cell beyond those inherent to the several varieties.

The intrinsic variations in the formation of the neuron are for the practical purposes of this examination reduced to the normal inequalities of the varicosities in the construction of the dendrons. These inequalities in the cortical dendrons are of comparatively infrequent occurrence, in contrast to those of certain of the cells of the basal ganglia, and belong either to the finer dendrites of the irregular cells, or to the points of division of the dendrites, and are then usually of triangular form and small size, and may readily be excluded in an examination for pathological lesions.

Besides the swellings in the course of the dendrons, we have always to be on the look out to exclude certain processes of the neuroglia cells that traverse long distances in the cortex, and exhibit a pearl-string swelling in the course of the fibre. These nodal formations lie at perfectly regular distances one from another, and present the appearance of a double cone of small size. It is only when these fibres are detached from the cell body that they can give occasion to error. Another source of trouble to the observer is the occasional deposit of rounded masses of precipitated silver on the dendrons or cell body, but this may usually be cleared away without difficulty by a careful examination of the edges of the questionable mass with strong transmitted light, when the irregular structure of the precipitate and serrated edges will at once appear. Still another source of error is the fracturing of the protoplasmic processes by the sharp edge of the knife (see fig. 4), the potash salt rendering the protoplasm brittle, but once aware of the difficulty it is easy to eliminate it as a source of error.

After all possible allowances have been made for artefacts and physiological variations, together with inequalities in the staining process, there remain a large number of cells in the alcoholic brains that are distinctly abnormal. The principal lesions to which we would call attention are a distinct diminution in size, a shrinkage of a vast majority of all the cortical cells, disappearance of the gemmulæ, certain swellings in the dendritic processes; and roughening of the stronger processes, and to some extent of the cell body.

We have not succeeded in staining the fusiform cells satisfactorily in the alcoholic rabbit's brain, but with this exception the cortical cells of all the layers may in chronic alcoholism be considered to be involved in a process of retrogressive metamorphosis, some in greater, some in less degree, but all are implicated to some extent. The small cells of the outer layer show degeneration, the pyramidal cells, particularly the long ones, show the same changes in greater intensity, the lowermost pyramidal and irregular cells are equally involved. Fortunately the pyramidal cells, from their very fixed character and larger size, are more easily studied than the others, and our principal attention has been directed toward them.

It is extremely difficult to determine in the silver preparations the approximate proportion of cells that are normal, and cells that may definitely be held to be abnormal, from the fact that an extremely small proportion of the total number of cells in all the layers are at any time stained. In some of the preparations from the same brain but one in a dozen cells show any definite change, while in other sections nearly all the cells are altered. It would perhaps be fairly safe to conclude that one out of every three or four cells in the brains of the three alcoholic rabbits we examined show departures from the normal in some form, though the percentage is probably higher. Another source of error in estimating the numbers of diseased cells arises from the circumstance that we can form no idea whether the silver salt preferably impregnates the normal or abnormal cells, for even in histological preparations no one has advanced any plausible theory why the silver elects here and there a cell to impregnate, and leaves the others perfectly untouched by the salt. Golgi (*Berl. Klin. Woch.*, No. 14, 1894) himself opened the question as to the capability of the diseased cell receiving the silver stain, but could only answer it to the extent that even much degenerated cells were impregnated.

A careful comparison of a control preparation, and one from an alcoholic rabbit will show the following differences: the cell bodies and main processes are more even and smoother, the dendrites are broader, the gemmulæ are longer and more numerous, thicker, more feathery, apparently spreading over more lateral surface, and are very regular in appearance, while in the alcoholic there is a gap here and there as if some of them had fallen off, or had disappeared. Indeed, the difference between the two cells may shortly be expressed in that the whole aspect of the normal cell is stouter than its pathological fellow.

Among the cellular layers—and we take the pyramidal cells of all sizes as our chief exponent—we find a vast number of the cells which show upon their protoplasmic extensions one or more swellings of a rounded or elliptical form (figs. 3, 6, 7). Some of these tumefactions are very small, and only to be noticed after close search, while others are so large as to immediately attract attention, and excite suspicion that they are artefacts, and indeed it was only after a thorough examination by means of the electric light that we were able to disabuse ourselves of the idea that they were not artificial productions (fig. 5). Under the intense illumination of the electric lamp they appeared as uniform reddish bodies divided longitudinally (under high magnification) by a series of fine lines converging upon the substance of the unthickened portion of the dendrite. The number of the swellings upon an implicated dendrite may vary very considerably; a dendron may be almost wholly filled with the swellings, at other times there may be only one or two, of either large or small size (figs. 3, 4, 7, 8, 9).

The process of tumefaction always appears to begin at or near the fine free extremity of the dendron, be it the extremity of the main apical process or one of its collateral branches, and not infrequently the free termination of the dendron is seen to be somewhat swollen before any other portion of the cell is involved. Looking over the preparations a little closer, we find numerous cells with moniliform swellings stretched along the finer apical processes (figs. 3, 4, 6, 8, 9, 10), until a number are seen with the whole of the apical processes thickly studded with the bead-like swellings. Somewhat remarkable to relate, the basal dendrons are only exceptionally involved in the tumefactive process, though they do not escape alteration when the cell corpus eventually begins to shrink (figs. 10 and 12).

An essential accompaniment of the tumefaction of the protoplasm of the dendron is the loss of the gemmulæ. The smallest swelling causes along its edges an evident decrease in the number of the buds (figs. 4, 6, 7). After the swelling has increased to a considerable degree all disappear, while in the intermediary stages every step toward decrease and disappearance may be found. Between the points of swelling they are retained, but are often seen to be thinner and situated at more irregular intervals than normally. When long thickenings occur in the dendritic stems the buds are invariably lost. The last of all the gemmulæ to disappear are the thicker branched ones that project at intervals some little distance beyond the others, and at times, though very rarely, they may still be seen clinging to the sides of some not far advanced swelling.

A very accurate comparison, so far as outward appearances go, may be made between the moniliform swellings of the dendritic processes of the nerve cells, and the swellings on the branches of some species of the oak tree caused by a parasitic insect. Both have the same knots and swellings on their branches, even to the falling off of the finer lateral twigs on the tree when the disease has advanced to a considerable extent, and both may go on to produce a shrinkage of the substance, finally ending equally in decay and death to the plant and cell.

As the arterial changes found were comparatively unimportant in their character it would be difficult to ascribe this widely extended and curious process of tumefaction of the dendrites and disappearance of the gemmulæ to nutritive changes solely from a defective supply of nourishment to the nerve cell, though truly the definite enlargement of the peri-vascular spaces would indicate that there had been some previous disturbance in the circulation of the lymph currents of the cortex. It would seem much more plausible to attribute the lesions to the direct action of the poison upon the protoplasm, though why it should take this peculiar form is difficult to determine. The alterations can hardly be of fatty nature, as the bodies of the unstained cells show nothing of this nature beyond the ordinary amount of yellow pigment granules which in the rabbit is minimal.

It would appear from our specimens that the moniliform swellings of the dendrites and the loss of the lateral buds was the first step in a pathological process, which eventually was to end in the partial or complete disintegration of the cell structures, and with it the annihilation of the nervous forces produced by the cell, the latter probably preceding anything like cellular death, for clinically we frequently see almost complete dementia alcoholica, when according to the ordinary methods of staining, but comparatively few of the nerve cells show characteristic signs of degeneration.

Only a few of the nerve structures of the rabbit's brain give any evidences of deterioration of the substance of the corpus, beyond those already described, though occasionally a neuron may be found which exhibits a more advanced degree of degeneration. Then the processes immediately adjacent to the body are roughened and seamed like the coarse bark of a tree; the staining of the altered protoplasmic substance is irregular, the cell body is irregularly shrunken, the basal dendrites no longer stretch over wide areas like their normal fellows, but have dwindled, the gemmulæ are lost, and the processes are not stained for any distance from the corpus of the cell, but soon end in needle-like points (fig. 12). Some of these cells might even be described as being corrugated, from the very roughened appearance of their exterior.

The nuclei of the nerve cells are seldom to be seen in silver preparations; occasionally a vacuole, occupying the spot where the nucleus usually lies, can be determined, and in its centre a black dot, probably the nucleolus, may be found if a careful search be made for it, but no details of the intimate structure can be determined. A very few of the sections exhibit a process we have thought possibly may arise from defects in the impregnation, though on any ground it is difficult to account for. The dendrons and axons of the neuron are nearly perfect, while the cell body looks as if a portion of its substance had been scooped out down to the region of the nucleus. Such a cell is pictured in fig. 11.

The axis-cylinders of all the cells of the cortex were made the object of careful study, but no morbid alteration could be made out in them; but this continuance of the axon, when the other portions of the cell structures are far degenerated, is one of the peculiar features of the neuron which has before attracted attention, especially by Golgi (*loc. cit.*), who found that the axon resists to a greater extent degenerative processes than the dendrons, and may be found intact when the cell is reduced to a mere stump. In all our preparations very numerous axis-cylinders were stained, and could be traced from their commencement at or near the basal end of the cell, downward into the white layers, or upward toward the uppermost layer of tangential fibres; but no trace of unusual varicosity or break in their continuity could be discovered. The collaterals oft-times could be followed considerable distances, occasionally to their free endings, but nothing abnormal could be seen. Cells in an advanced stage of degeneration had as perfect axis-cylinders as normal ones, and could be traced equally long distances.

Cortex of the Cerebellum.

The Golgi cells and the Korbzellen of Koelliker stained only in inconsiderable numbers, and but few of those impregnated showed changes comparable to those of the cerebral cells. The Purkinje cells, on the other hand, were luxuriantly tinged, sometimes an entire row being blackened and exquisitely brought out. A better comparison could therefore be made in these preparations between the numbers of degenerated and undegenerated cells—the proportion now being, when an entire row was taken, one degenerated to three normal ones.

Practically the same alterations were found with the Purkinje as with the pyramidal cells, though the changes were more striking from their relative intensity. The loss of the lateral buds, from their greater luxuriance, was extremely striking; they disappear from the dendritic branches as an entirety, and on a considerably degenerated nerve body not a single one can be found (figs. 15, 16, 17).

The stems of the dendrites also undergo atrophy, and in great measure disappear; only thick stems are now seen arising from the corpus of the cell, which give off a number of short, stumpy branches, thickly studded with knotty projections in the place of the long feathery dendrites and their terminal twigs (fig. 14). These twisted and shortened stems have numerous swellings in the longitudinal direction of their protoplasm. Those that still retain something of the form of a dendrite have numerous irregular swellings in the stems of the finer twigs; the gemmulæ are almost universally lost, and the whole appearance is as if the dendrites had become gnarled and knotted by the action of some parasitic insect (fig. 15). The cells that are least implicated have the rounded and longitudinal swellings along their finer stems, and the buds along the margin are fewer than elsewhere.

The degeneration of the Purkinje cell, while having the same general characteristics as the cell degeneration in the cerebrum, is much more striking, and there is no possibility of mistaking the process for anything else than it actually is—a degeneration of the cellular structures.

Neither in the cerebrum nor cerebellum is there any pronounced change in the support elements. The Deiter's cells stain in neither region with any frequency, while the large-bodied vascular cells do not seem to take on any alterative action, clinging to and surrounding the vessels after the usual manner.

Before concluding this paper let us glance for a moment at the clinical picture of chronic alcoholism in order to see in what manner the pathological lesions and the symptoms correspond with one another. The epiphenomena of chronic alcoholism are so numerous that it will be difficult to do more than sketch the most prominent symptoms: First come the sensory disorders, the exaggeration of the sensibility of the skin, the anæsthetic symptoms, the ocular and auditory troubles; then follow the tremor, the tremulous articulation, the loss of tone in the muscular system, the tottering gait, the occasional paretic attacks, the inco-ordination, and lastly the intellectual disorders, the loss of moral force, the weakening of the mental faculties, the falsification of the judgment, the slowness of memory, and finally the dementia, which is seldom complete.

The first of these periods would correspond to the beginning of the vascular disorders, when the nerve cells, irritated by the presence of a poisonous stimulus and an insufficient supply of proper nourishment, overact for the time, and then as nutriment is still withheld from them, altered metabolism results. The beginning swellings of the dendrites of the sensori-motor region is marked by anæsthetic and paræsthetic symptoms, those of the purer sensory regions by visual and ocular troubles and some amnesia, especially for recent events, or in other words, cells that have the function of evolving thought cannot work properly, and defective memory results. Later, as the motor cells become more and more involved and nuclear changes begin, continuous tremor becomes apparent, the muscles no longer co-ordinate perfectly, unless for a moment under the direct influence of the will; still later, when a portion of the cell structures become highly degenerated, and the altered cells have become more numerous, the already tottering will power becomes less and less active, judgment and memory fail, and when the degenerative process has far advanced an incomplete dementia is the final result.

In conclusion, we do not consider the fact that only a portion of the cells of the cerebrum are involved in the degenerative process militates aught against the entire conception of the pathological entity. The nerve elements of the brain are intricately united one with another by means of their axons and collaterals, and are not able to function completely unless the normal relations one to another are preserved. A lesion in one cell will induce disorders in the function of two or more cells not involved by any morbid change, the intricate system of collaterals issuing from one cell influencing directly the impressions and nervous forces arising from many others, and in this way a widespread disordered action of large numbers of the cerebral cells may be the result of disease in a comparatively few elements.

In the human being we have found the same degenerative process present as in the rabbit, varying only in degree, in some more intense, in others less so; but as the pathological lesions in chronic alcoholism in the human subject will form the basis of a subsequent paper, it will suffice here to say that they are practically identical with those in the rabbit.

We do not for an instant consider these lesions of the neuron peculiar to the effect of alcohol, but regard them as capable of being reproduced by any irritant drug or bacterial toxic product circulating in the blood, and acting for a considerable time on the living protoplasm of the nerve cell.

One point is made definite by this study—the fact that the alcohol which was supposed to be the least deleterious of all the series, exerts a very definite and destructive effect upon the nerve cell.

EXPLANATION OF THE PHOTOGRAPHS AND DRAWINGS.

Photos.

FIG. 1.—Nuclei of normal pyramidal cells of the rabbit's cortex, showing the nucleoli smooth and sharply defined. (× 1100.)

FIG. 2.—Nuclei of the pyramidal cells of the alcoholic rabbit's cortex, showing the nucleoli roughened and enlarged. The outline of the nucleus is also more irregular than in the normal cell. Rabbit No. 10. (× 1100.)

FIG. 3.—Beginning moniliform swelling of the apical process of a small pyramidal cell. Rabbit No. 42. (× 275.)

FIG. 4.—Moniliform swellings in detached portions of apical dendrites. Rabbit No. 42. (× 275.)

FIG. 5.—Large swelling in the apical process of a pyramidal nerve cell. The broken appearance of the dendrons in the figure is artificial and made by the microtome knife. Rabbit No. 41. (× 265.) Micro-photographs by Dr. A. G. Hoen.

Drawings.

FIG. 6.—Small pyramidal nerve cell from the outer portion of the second layer, with normal body and basal and apical dendrites, but with one branch from the apical dendrite showing moniliform swellings. The gemmulæ have disappeared wherever a swelling has begun. (× 560, enlarged.)

FIG. 7.—Small irregular nerve-cell from the outer portion of the second cell layer with advanced moniliform swellings of all the principal dendrites, and roughening of the cell corpus. (× 560, enlarged.)

FIGS. 8, 9.—Advanced moniliform tumefaction of apical dendrites. (× 560, enlarged.)

FIG. 10.—An irregular pyramidal cell from the deeper portion of the second layer in an advanced state of degeneration.

FIG. 11.—Pyramidal cell from the deeper portion of the second layer with slight roughening of the apical process and normal dendrites. The corpus of the cell is excavated from its basal edge to the further limit of the nucleus.

FIG. 12.—A medium-sized pyramidal cell showing disappearance of the long dendrites and great roughening of the thicker stems, as well as complete loss of the gemmulæ. The cell body has a very irregular contour and is shrunken. The axon is intact. (× 560, enlarged.)

FIG. 13.—Terminal portion of a dendrite of a Purkinje cell showing the normal arrangement of the gemmulæ. From a control preparation.

FIG. 14.—Portion of a process of a Purkinje cell from near the corpus. The longer branches and gemmulæ have entirely disappeared, leaving an irregular knotted stump. (× 560, enlarged.)

FIGS. 15, 16, 17.—Terminal branches of the dendrites of Purkinje cells, showing the irregular swellings of the stems, loss of the gemmulæ, as well as the finer branches. Rabbits Nos. 6 and 41. (× 560, enlarged.)

DR. BERKLEY ON THE CORTICAL NERVE CELL.

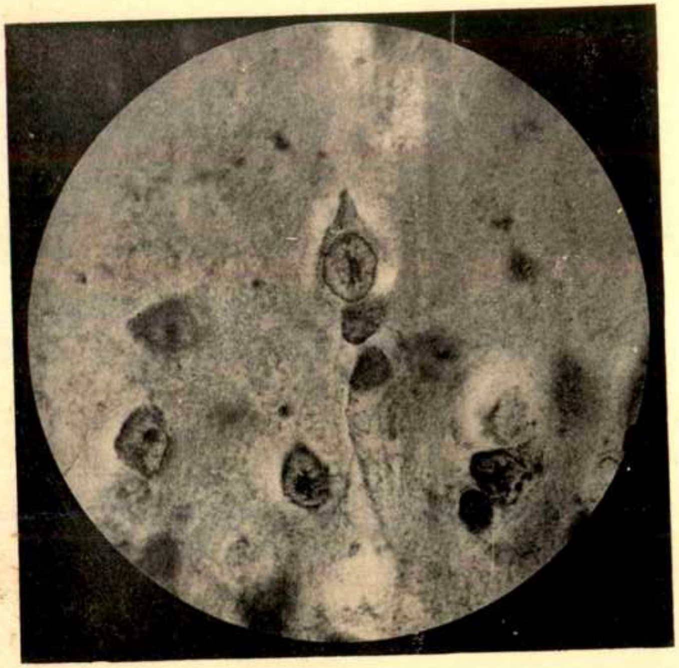


FIG. 2.

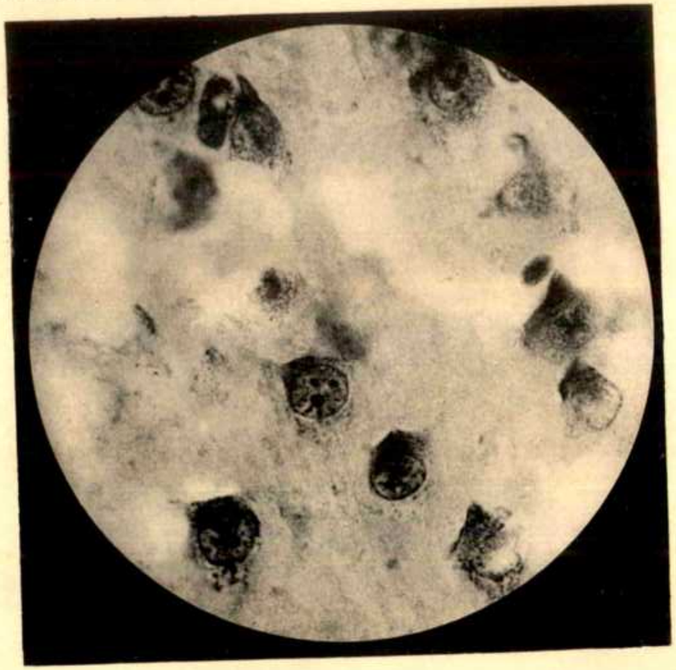


FIG. 1.

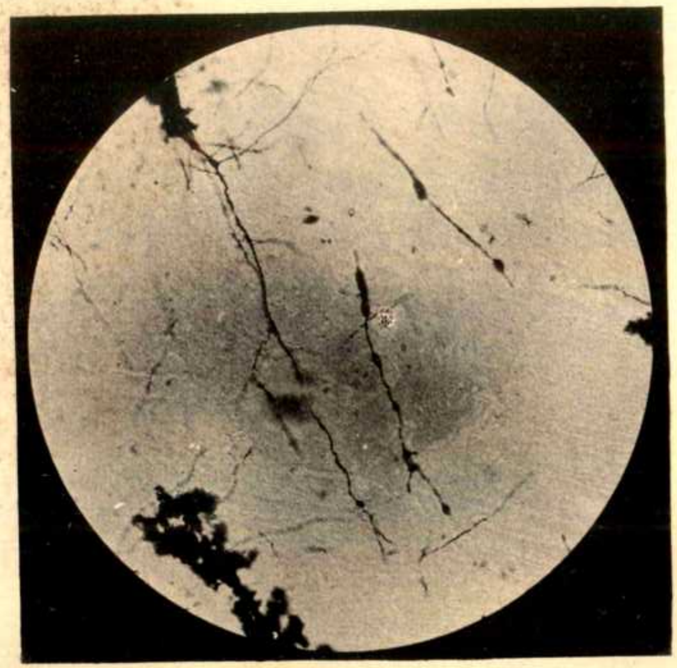


Fig. 4.

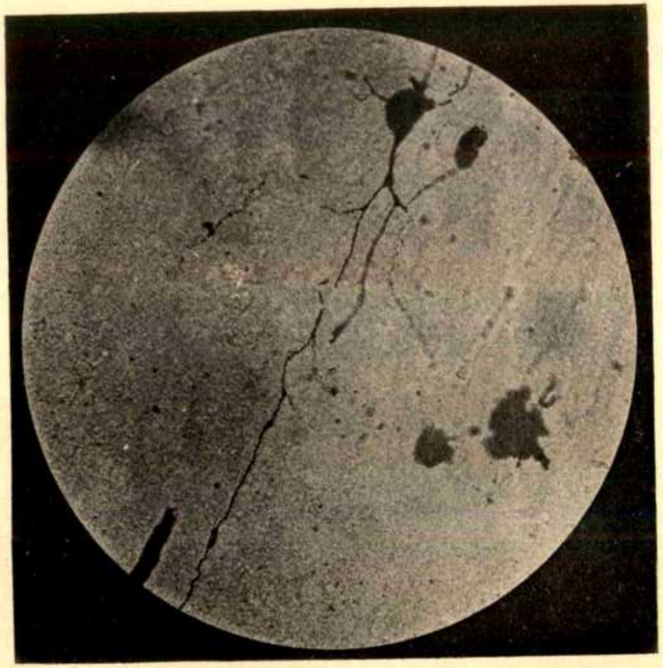


Fig. 3.

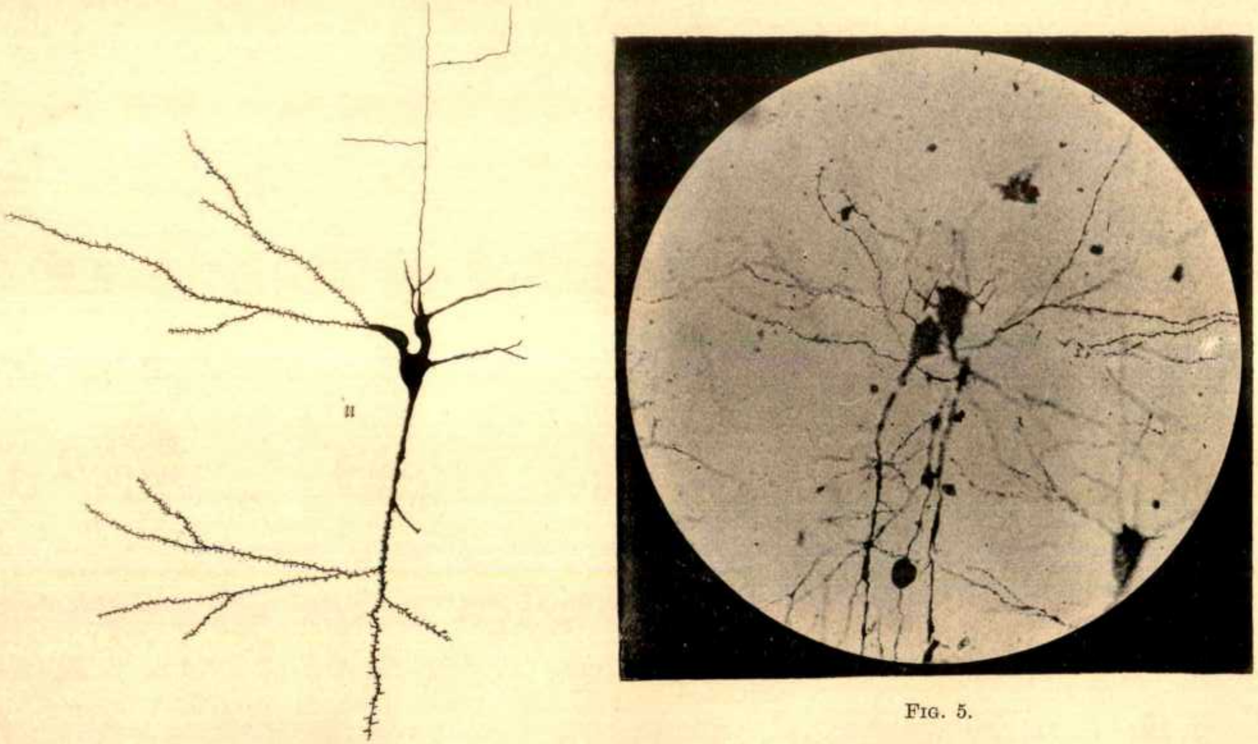
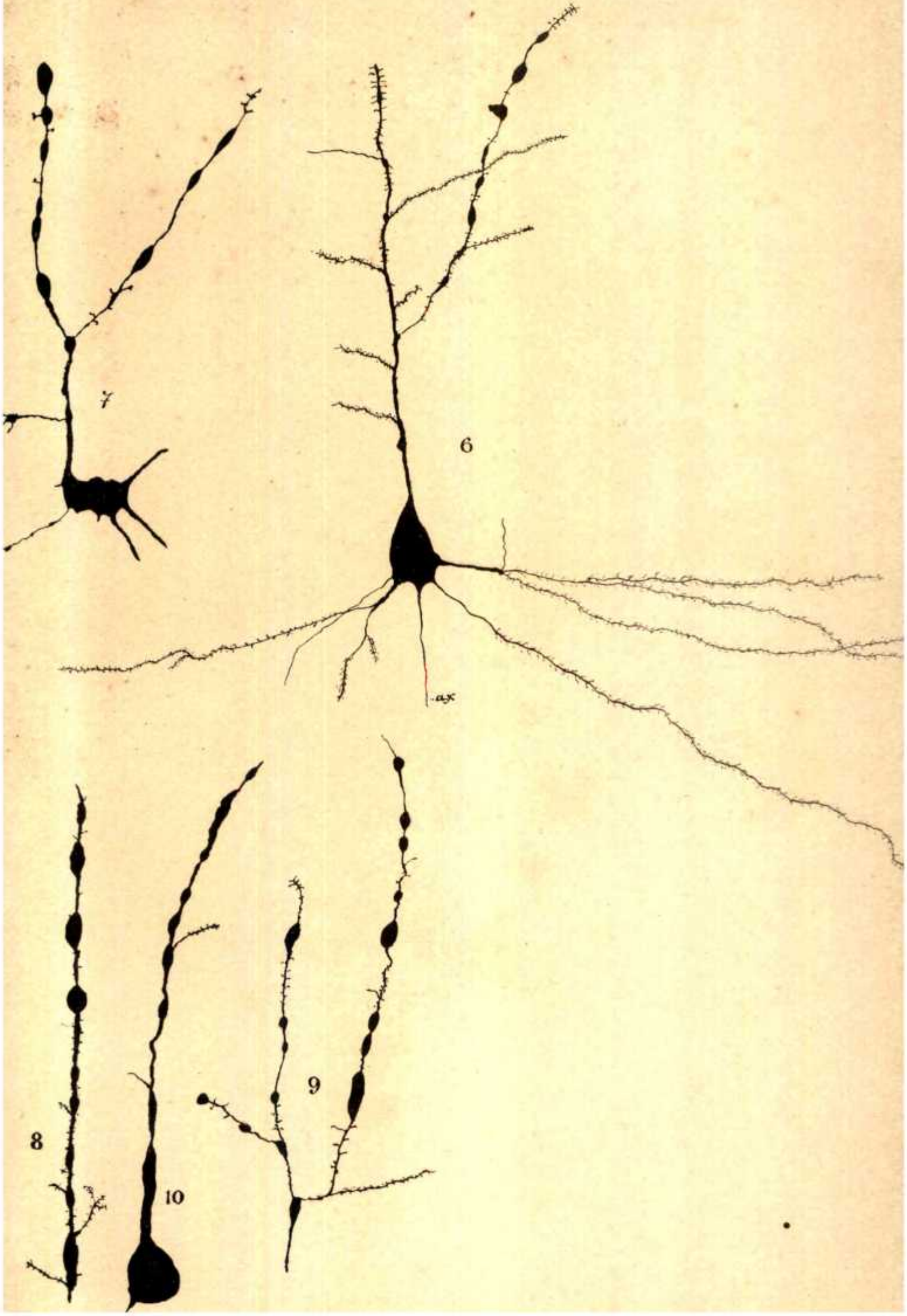


FIG. 5.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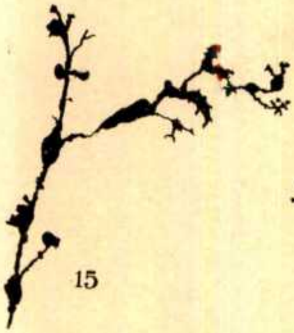

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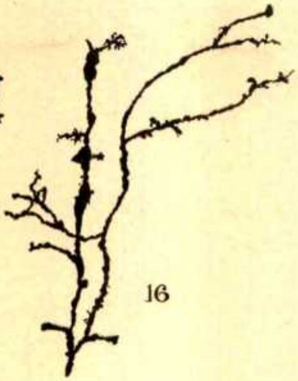

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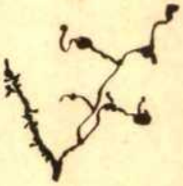

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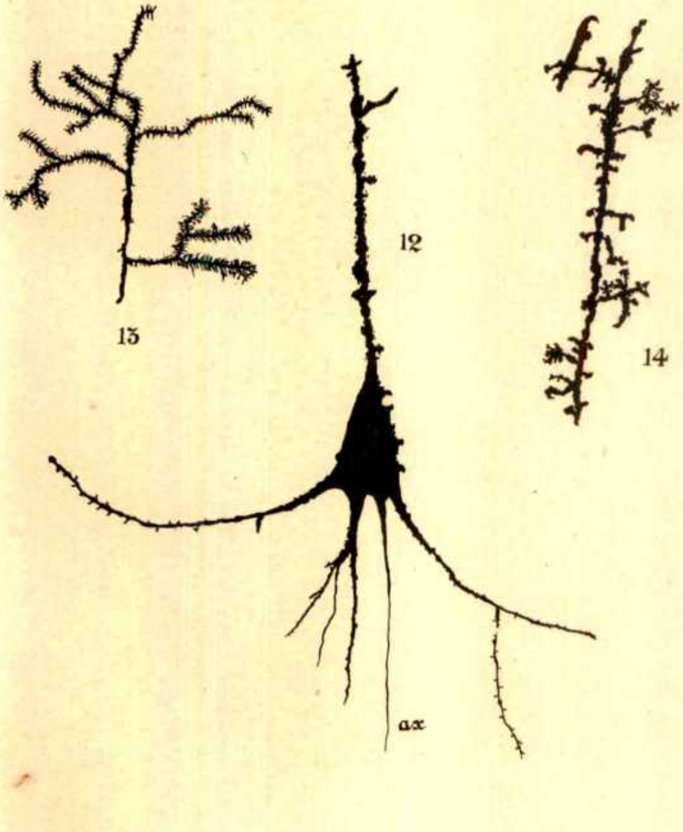

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THE FUNCTIONS OF THE FRONTAL LOBES.

BY L. BIANCHI, M.D.

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[*Translated from the original MS. by A. de Watteville.*]

THE experimental problem raised by the question we are about to discuss is of recent origin, and aroused the attention of observers soon after the discovery of motor and sensory centres. On electrical examination of the cortical surface, the anterior frontal lobes were found not to be excitable; and whilst ablation of other areas was followed by more or less evident loss of movement or sensation, that of the præfrontal region gave rise to but slight, or doubtful, disturbances.

These negative results are the cause of the small amount of attention paid to the physiology of the frontal lobes, as compared with the activity displayed in experimentation on the motor zone. But I hope that the experiments I am going to describe, conducted on those more rigorous lines adopted by our modern science, will, to a certain extent, make up for the deficiencies of the previous results. I think, however, that we may pass over the researches of Müller, Longet, Vulpian, Lussana, and others; they vainly strove to transfer into the domain of the positive school, the results of the reaction against Gall's organology, such as that intellective activities (images, memories, ideas) are not localised in any particular areas of the cortex.

The classical work of Hitzig[1] afforded a more secure starting point, and by new methods opened a new phase in the evolution of our knowledge of præfrontal functions. He observes that anatomically the development of the frontal lobes is found to keep pace with that of the intellect, being less in the cat, for instance, than in the dog, and still more so than in the monkey; hence the author concluded, naturally enough, that they are really the seat of the highest intellectual faculties. But Hitzig's experimental results did not confirm this very plausible argument. His dogs never showed intellective disorders, either after superficial lesions, or after complete extirpation, of the frontal lobes.

Ferrier[2] next attempted to solve the problem, on the strength of a more abundant experimental material. He assumes that all in front of the sigmoid gyrus represents the frontal lobes in dogs and cats; in monkeys, what is in front of the centre for the movements of the eyes and face and the opening of the lids and dilatation of the pupils. Electrical excitation of this region has always been negative, except in one monkey, in which ocular movements were observed.

Mutilation of this region gives negative results in dogs. Monkeys (three in number) showed no alteration in their movements, sensations, instincts, feelings, or emotions. There was, however, some alteration in the temper and behaviour of these animals, says Ferrier, though it is difficult to specify its nature. They took less interest in their surroundings; showed less of their natural inquisitiveness; were apathetic, somnolent, responded only to momentary impressions, and had lost the power of attentive observation. Ferrier attributed motor functions to the frontal lobes on account of their connection with the corpus striatum; they are inhibitory centres, whose activity is exerted on influencing the actual motor centres. Inhibition thus understood is nothing but attention, which is thus to be considered as a function of movement. Hence his conclusion that the frontal lobes are the centres of attention.

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[1] "Untersuchungen ueber das Gehirn," 1874.

[2] "The Functions of the Brain," London, 1876.

Ten years later, in his second edition, this author comes to nearly identical conclusions. He confirms the absence of electrical excitability of the part of the frontal lobe placed anteriorly to the eye and head centre (No. 12 of his diagram). He relates new experiments on the destruction of the præfrontal region, remarking, concerning one of them, that notwithstanding the bilateral lesion, there is complete absence of symptoms, either motor or sensory. The animal feels contact over the whole body, the special senses are intact, and the movements of the head and eyes are completely preserved.

Extirpation of the anterior extremity of the frontal lobe having produced the same deviation of the head and eyes as that of the region immediately behind (marked 12 in the diagram), Ferrier was led to consider the two as essentially the same functionally, viz., as motor of the head and eyes.

It is impossible to anyone familiar with these experiments not to look upon this conclusion as premature. Ferrier was too much impressed by the phenomena observed immediately after the operation. We now know how many errors are committed by this cause, as Goltz pointed out long ago. Nor does the argument derive much support from the fact of the anatomical relationships shown to exist between the frontal lobe and the pons and medulla by the microscopical examination of brains thus operated upon. For nothing proves that a centre of higher function cannot have relations with the pons, cerebellum and medulla; and that a degenerated tract traced through the internal or mesial part of the internal capsule and pes cerebri must necessarily subserve movements. And even if the frontal lobe were motor, it does not follow that it must be considered as the centre for the head and eyes, as are the roots of the superior and middle frontal convolutions.

This criticism is the more founded, as we find that in one of the experiments, destruction of the frontal lobes gave rise to no symptom, though secondary degeneration was traced through the internal capsule.

Even in this second series of experiments, however, Ferrier succeeded in showing evidence of a mental deterioration. The monkeys, after the operation, had lost the power of attentive observation. But this fact was much less evident when, through antiseptic precautions, the animals were enabled to survive for a long time. At any rate, as the author says, this fact was more apparent than real. But, adds Ferrier, the frontal lobes are not the seat of intelligence; this would be an absurd statement, since intellect and will have no distinct localisation, but are the resultant of the functions of the whole cortex, and are correlated to the harmonious development of its various portions.

Thus Ferrier conciliates his first hypothesis, that the frontal lobes are the centre of attention with the views promulgated in his second edition, according to which it is so because it is a motor centre, and makes part of the motor zone.

Moolmann's researches added nothing to the doctrine propounded by Goltz, in whose laboratory his researches were conducted. Here are his conclusions: disorders consecutive to destruction of one frontal lobe do not differ as to persistency from that of any other part of the cortex; partial destruction of the frontal lobes causes but insignificant symptoms. When both frontal lobes are completely removed, the consecutive disturbances are the same as those observed after ablation of the parietal lobes, which means, according to Goltz, of any other part of the cortex.[1]

Goltz[2] so far recognised that the animals deprived of their anterior lobes, without being actually paralysed, cannot bring into play the complex processes of innervation necessary to the normal performance of actions, and lose their power of voluntarily moderating their bulbar and spinal reflexes.

Hitzig returns to the subject, and by new experiments on dogs, combats Munk's theory, and asserts that ablation of the frontal lobes is never followed by any motor disturbance.[3]

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[1] *Pfluger's Archiv*, Bd. xx., 1879.

[2] *Ibid.*, vol. xxxiv.

[3] "Achte Wanderversammlung d.S.W.D. Neurologen," *Neurol. Centralbl.*, 1883.

We now come to Munk's researches, which were favourably received in Germany, with reference not only to frontal functions, but also to the sensorimotor localisations of the cortex generally. He used dogs and monkeys. Here is a summary of what he says of a dog: Three to five days after a unilateral operation the animal is as lively as before. It manifests no disorder as to vision and hearing under any test or circumstance. It sees, hears, tastes, understands when called. Internal sensations are normal; likewise general sensation and intelligence. A dog thus mutilated cannot be distinguished from a normal animal. It walks, runs, jumps, moves its eyes, ears, tongue and tail as before the operation. It barks, bites, feeds as before. The movements of the trunk alone show some disturbance. If the extirpation has been carried out on the left side, the dog can walk and turn to the right and to the left. But when it turns suddenly it forms an arch, whose convexity is towards the right side, the body is arched on the left, the vertebræ forming a convexity to the right. On running it can describe large curves to either side, but sharp curves are possible to the left only.

Thus far Munk. Extirpation of the left frontal lobe causes nothing but slight trunk paralysis, of long duration, persisting for months. Extirpation of both lobes produced an abnormal arching of the dorso-lumbar spine; especially on walking slowly or rising from the ground the animal displayed this symptom (cat's back), but not on running or jumping. No disorder of intelligence nor of the various forms of sensation were observable, whenever the excision involved the frontal lobe lying in front of the chief frontal sulcus (Hauptstirnfurche). Similar results were obtained in analogous experiments on monkeys. After the direct effects of the operation had passed away, the animals deprived of their frontal lobes lost their smell only when the olfactory lobe had been also injured. The trunk muscles alone were affected; no disorder of sensation, sensibility, nor intellect was noticed. The animals could not bend their dorso-lumbar spine to either side in the performance of the smaller movements. They could not straighten the back, which remained inflected, with approximation of the anterior and posterior limbs. What Ferrier observed (apathy, somnolence, unrest) is, therefore, attributed by Munk to the effects of the lesion itself, to encephalo-meningitis.

Munk, therefore, looks upon Ferrier's experiments as failures, denies the existence of any loss of attention, perception and thought, and states that there is no failure of that inhibition which Ferrier takes to be the physiological substratum of attention. He found that a monkey so operated was kept, by a mere look or gesture of menace, from touching a much coveted morsel of food placed before it. This monkey used to take a piece of carrot held between the thumb and index, but not between the thumb and middle finger until the index had been drawn back.

Luciani[1] reports only two experiments on dogs which were deprived of one frontal lobe only. In both cases he observed nothing but a paralysis of the dorsal muscles and sensory troubles, with paralysis of the anterior limb, ascribed by him to a lesion of the anterior limb of the sigmoid gyrus.

The researches of Horsley and Schafer[2] are not at all conclusive, because the mutilation was confined to the anterior third or fourth of the frontal lobes. None of the seven monkeys showed any disturbance of motion, or special sensation nor intellect. These results are wholly contradictory to those of Munk, and partially to those of Ferrier, who in a recent publication,[3] restates his former position without any modification.

Lastly, Grosglik[4] has just published a paper, in which the bibliographical references are incomplete. His experiments have been performed only on dogs, and are therefore undecisive on such a question. He reaches the same conclusion as Munk in so far as the frontal lobes are the common centres of the neck and back, but on the other hand he maintains that the motor troubles of the spine are not persistent.

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[1] Luciani e Seppilli, "Le localizzazioni funzionali del cervello," 1885.

[2] *Philosophical Transactions of the Royal Society*, 1888.

[3] "Croonian Lectures," 1890.

[4] Grosglik, "Zur Physiologie der Stirnlappen," *Archiv fur Anatomie u. Physiologie*, 1895.

Let us now summarise the various opinions hitherto propounded on this subject:

(1) The præfrontal lobe is the motor centre of the eyes and head on the opposite side; and in consequence of the close relationship between the movements of these parts and attention, it is also the centre for attention.—(Ferrier.)

(2) It is the centre for the highest psychical functions. Destruction of it involves a real decadence of psychical activity.—(Wundt, Hitzig, Bianchi.)

(3) It is part of the so-called "Fühlsphære," and as such is the motor centre of the dorsal muscles. Its highest development is not correlated with that of intellect, but with that of the dorsal musculature.—(Munk, Luciani.)

Such were the various hypotheses concerning the frontal functions when, with wholly unprejudiced mind, I began my own researches in 1888. I have been prosecuting them ever since, making short communications to the Royal Medico-Chirurgical Academy of Naples (1889), to the Society of Italian Alienists (1890),[1] and to the Congress of Internal Medicine in Rome (Oct., 1892). My experiments were made on monkeys and dogs, after the method of free cranial openings and exposure of the lobes, rigorous asepsis and observation of the animals for several months or even years. I took no account of the symptoms during the first week, which must be systematically overlooked in researches on the cortical functions by the method of extensive mutilation.

I used twelve monkeys and six dogs, and never lost an animal immediately after operating, even after total extirpation of both frontal lobes. I lost a monkey in my laboratory at Palermo, from consecutive hæmorrhage; and one last year, a rickety subject, with a thick skull that offered great difficulties in operating. In determining the præfrontal zone, I have not, as did Munk, taken the sulci as landmarks, first, because they never demarcate the physiological areas of the cortex; secondly, because even if such were the case the experimenter cannot always strictly follow their indications.

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[1] See abstract in "Untersuchungen z. Naturl. d. Menschen u. d. Thieren," *Moleschott's Arch.*, vol. xiv.

Hence I have not always hit upon the præcentral sulcus in monkeys, nor upon the extremity of the chief frontal sulcus (sigmoid fossa) in dogs.

I have taken as my chief guide the electrical reactions of the frontal region, exploring it repeatedly with an exciter connected with a du Bois Reymond's sledge apparatus. I used to test the strength of current on my tongue, always keeping to the same intensity of sensation, which corresponded to 9 or 10 mm. of the scale. I avoided the use of the galvanic current for fear of trophic alterations in the cortex. On account of the slight individual differences in the motor localisations, especially in the monkey as compared with the dog, I thought it best to carry out the incision two or three millimetres in front of the excitable areas of the arm, face and jaw. I thus almost always in monkeys hit upon the areas, excitation of which produces dilatation of the pupil and lids, and turning away of the head more than of the eyes.

On account of the obliquity, from below upwards, of the Rolandic fissure, it is evident that a greater portion of the upper escaped, than of the middle and lower frontal convolutions.

I did not make any special efforts to spare the olfactory bulb in monkeys, in whose psychical life it plays but a subordinate part; but I always preserved it in dogs. In these animals the line of section fell at less than a millimetre behind the sigmoid fossa; the anterior arm of the sigmoidal convolution being almost always involved.

I shall not dwell upon the results of the electrical excitation of the zone thus removed; they mostly agree with those of other observers; the surface of the frontal area situated in front of the motor area of the head and neck, and of the iris is quite unexcitable with currents of equal strength. I shall only remark that such an excitation of the foot of the upper frontal convolution produced (in monkeys and dogs) not only a slight rotation of the head to the opposite side, but also some lateral displacement of the trunk at the level of the lumbar region.

Conjugate movements of the eyes did not always accompany rotation of the head. Excitation of the inferior area produced raising of the eyelid along with dilatation of the pupil. In one case this dilatation in the opposite eye could be observed fifteen minutes after the stimulation, which, as I have already stated, I used only in order to fix the line of incision. The latter was not identical in all experiments, on account of individual or of operative differences, hæmorrhage or adynamia compelling me occasionally to hasten the operation. Three to five days after the operation the animals were fit to undergo all the investigations required to determine functional losses.

The following statements summarise the results obtained by unilateral extirpation of the frontal lobe.

(*a*) During the first and second week I often observed, in monkeys oftener than in dogs, rotatory movements, the truncal concavity being towards the mutilated side. No concomitant oculo-motor disturbance is observed. These symptoms do not always occur, and rarely last beyond the second week.

(*b*) Paresis of the opposite arm, not evident in associated movements (climbing, seizing a stick, shaking objects), but obvious in more delicate movements, when the monkey uses the other hand (for picking up a fruit or other food, catching fleas). The animal then uses the paretic only when the other is immobilised. This paresis also disappears after a few days—three weeks at most. In some of the subjects it has not been observable, or only slightly so.

(*c*) Tactile sensibility remained normal, except in a dog, in whose opposite limb it was for a time distinctly diminished, and in a monkey who showed some hyperæsthesia in the opposite ear and face. All the animals scratched themselves equally on both sides, and the monkeys hunted for fleas with their usual and characteristic precision.

(*d*) In one case (a monkey) I found diminution of hearing on the opposite side.

(*e*) Taste appeared normal, as well as smell, so far as could be ascertained in so difficult an investigation.

(*f*) In all cases, monkeys and dogs, I noted visual disturbances. I shall quote the observations made in one instance:—"The opposite eye being closed, the monkey walks, runs, without ever coming in contact with any of the obstacles placed in his way; he avoids them all without uncertainty. But let him be kept immobile, and his attention be directed on fruit held at from 30 to 40 cm. in front of him; if then a piece of sugar, of which he is extremely fond, be suspended by a thread and gradually brought from the right inwards into his visual field, the animal sees it only when the sugar is nearly in a line with the visual axis. It is only then that the animal perceives it, and seizes it with a rapid and precise movement of the left hand. This does not occur when the right eye is kept closed; as soon as the sugar passes the physiological boundary of the visual field the animal's attention is excited, and the action of seizing the object is instantly performed. This experiment never failed on repetition; the difference between the two eyes is striking. The results were the same a fortnight, as they were a week, after the operation. Generally, these visual disturbances last from two to three weeks.

No perceptible differences were noted in the behaviour or psychical manifestations of animals mutilated on one side only.

I shall now give some details concerning the psychical condition of one dog, and of three monkeys operated bilaterally.

(*a*) Dog whose two anterior lobes were removed on April 16 and on May 16 respectively. The sensory and motor functions of the limbs remained normal. On June 21 the following notes were taken:—As soon as the door of the room in which it is confined is opened, the animal walks out. It goes with eyes downcast, and head bent down, almost touching the floor with its nose. It hovers about heavily, aimlessly; picks up in its mouth all what it finds (leaves, branches, filth, &c.), dropping it out again. If its way is barred with a hat, it is frightened, stops, but does not try to turn the obstacle. On finding a basin in the middle of an alley in the garden, it turns round about it excitedly, first in one sense, then in another, and so on. It starts impulsively to run, but gets entangled in the chain of another dog (amblyopic through occipital mutilation), and gets bitten. Frightened, it doubles itself up, without any attempt to escape from the attack. On being helped out by the attendant it begins to run about furiously, avoiding obstacles; but gets entangled and bitten again, and howls desperately until helped again. The animal, however, eats, drinks, and recognises its kennel.

(*b*) Chacma (Cynocephalus Porcarius). Adult intelligent female. Walks on all fours, or on the posterior limbs only, to command. Renders military salute, seizes and drops a stick, understanding words without gesture. Quick at new spontaneous adaptations; *e.g.*, raising and dropping the latch of a door through which she wants to get out. Distinguishes various foods, picking out without hesitation the preferred kinds. Observant and friendly; showing affection to another male cynocephalus recently operated, but without any erotic tendency even during the menstrual period, taking him in her arms, almost kissing him, pressing him to her breast with a satisfied and almost ecstatic look. This expression is still more evident in the case of two puppies, which she adopted from the first, and treats in a motherly way. These are the notes taken six days before the operation:—

May 23, 1892.—To-day a puppy accidentally got near her. She first looked at him with surprise and curiosity, then took him up gently on one arm, caressing him, and showing all the care and devotion which a mother shows to her child. She put him on the floor, followed him about as far as her chain would allow, gently picked it up and carried it back; then, fearing to lose it, she would keep it in her arms, or on her lap with one arm passed under the chest. Nothing induced her to let it go. If, when the puppy was on the floor, one called or whistled it to come, she understood the signal at once, picked it up and kept it close to her body. If one threatened her with a stick or otherwise, she became frantic, holding the puppy firmly but yet tenderly, and climbing to the top of the grating for safety. With great difficulty it was removed from her, but was restored soon after. She showed great pleasure, and as if grateful to me, came several times near me, holding out her hand for a caress.

A second puppy was placed near her, and began to prowl around She examined, touched and sniffed it, but did not pick it up. But, when it began to move away, she seized it by the tail and pulled it to her; this was repeated several times. When she moved about she kept holding one of the puppies under one arm, and pulling the other along by the tail with the other hand.

It was impossible to get them away from her; and when anybody came near, she held both, each with one arm, and endeavoured to place them out of reach. In order to make her release them it was necessary to resort to a jet of water, of which she has the greatest horror, the very sight of the apparatus throwing her into the wildest excitement. Now, in spite of the jet, she for a considerable time stuck to the two puppies, trying to shelter herself anyhow. At last she dropped the second puppy and climbed to the top of the grating with the other under her arm. Tired out at last, she gently deposited it on the ground and again climbed to the top.

May 29.—Operation on the left frontal lobe. About $3\frac{1}{2}$ grammes of cerebral tissue were removed, occupying over 2 sq. cm. in front of the foot of the first and second frontal convolution. Slight hæmorrhage; strict antiseptic precautions.

The next day she was feverish in the morning; but the temperature became normal in the afternoon. She was rather in a low state, but still could be fed.

June 2.—Right paresis. She uses the four limbs to walk, but is strong enough not to fall on the weakened side. In climbing, she uses the left limbs as chief points of support; the right hand, is stronger than the corresponding arm, which affects a paralytic posture during rest. Even with threats one does not elicit the military salute; she cannot pick up objects with the right hand, and when made to use it, she makes great efforts, and the object easily falls from the hand; she cannot carry it to her mouth. The posterior limbs are flexed; she cannot, as before, stand upright on her feet.

The external segment of the visual field is much enfeebled in the right eye; the nature of objects presented is not recognised therein, as it is in the internal segment. The experiment is decisive when a cherry is offered to her.

June 5.—Walks well; no "mouvement de manège." Does not use the left hand, only as when a cherry is seized or she tries to open a door, or raise herself to see what is on a table. She no longer gives the right hand, nor salute (*i.e.*, stands on the hind legs, seize the tail with the left hand, and carry the right to the forehead military-wise). The diapason is heard on both sides equally. The cherry is clearly perceived only on arriving from outwards to the middle line, or visual axis, of the right eye. She scratches the right side of the head and face with the right foot or left hand.

She is still susceptible of feelings and new adaptations. Her favourite puppy being offered, she takes it up, hugs it tenderly with the left arm only. She hunts for fleas with the teeth and left fingers; and resents any attempt to remove the dog. She cannot climb on a chair with the puppy; she places it on the chair first, and climbs afterwards. She did not care for, and avoided, the second puppy. Out of four she cares only for the one she had previously taken to, and keeps it closely when they are called away.

June 26.—The paretic limbs have been recovering stength; for fine movements she still prefers the left hand; when compelled to use the right she is still uncertain. Vision is not yet perfect in the affected region.

August 16.—Second operation on the right frontal lobe. The trephine is applied to the point corresponding to the root of the frontal convolutions. Faradic excitations are followed by contractions of the left limbs, face, and peri-cranium respectively. Movements of the ear alone can be elicited by excitation of a point near that where movements of the peri-cranium and ear are obtained. About four grammes of frontal brain substance are removed. The incision falls accurately in front of the first and second frontal convolutions; and all the substance so separated is carefully removed. Much hæmorrhage. Two hours after the operation the animal drinks a glass of milk.

August 19.—Considerable swelling over the seat of the operation; no fever. The animal stands on its hind legs, but the left gives but uncertain support. On walking on all fours, the left arm drags. Tendency to rotation to the right.

Aimless instability of action. Reflexes exaggerated on both sides. Moderate excitations (by touching) produce generalised disorderly spasms. Left eye almost completely blind.

August 24.—Less rotation to the right. Still much instability; the animal climbs up and down a chair placed near, upsets it, pulls and shakes it about, opens and shuts a grating, pulls about her chain, and begins to rotate when not engaged in some of these actions. All this is done in a listless, automatic manner. She avoids impending falls or hurts, but immediately exposes herself again to the same accidents. She takes no interest in her neighbours and their actions, does not respond to calls, and manifests a quite unusual feeling of terror. She is even afraid of the other cynocephalus, whom she previously used to hug with evident pleasure. She has lost her previous interests, *e.g.*, in the puppy, of whom she is even afraid if placed near her.

Reflex hyperexcitability diminished. Slight hyperæsthesia on the left side. The right ear seems more sensitive to the diapason than the left. In order to test taste, two similar pieces, of sugar and plaster respectively, were offered; she took the one nearer to her—the plaster—chewed it, and swallowed it, without apparent feeling of pleasure or pain. The same happened with the sugar, which she chewed and swallowed automatically. The experiment gave the same results a second time, when she again took the plaster without paying any of the minute attention to it as she used to do when any strange food was offered.

Vision was tested by different methods, and considerable impairment was constantly proved to exist. A fig, after being carried over a wide area of the external visual field of the left eye only, was placed close to its left hand, without the animal taking any notice of it; whilst it immediately made efforts to seize another fig placed at some distance on the right side. This absence of perception was noted in all meridians of the visual field of the left eye; the object was seen only on entering that of the right eye.

October 20.—The same automatism and semi-consciousness are still observed in all the psychical and motor manifestations of the animal. Rotation to right still considerable. On a loud call she shakes herself; and, after much practice, she attempts the military salute; and shows some feeling when caressed, and avidity when offered some food, but only for a short time together. The habitual state is still one of indifference; and she speedily relapses into an aimless, automatic mode of life, without any interest in her surroundings. Under novel conditions she is not capable of new adaptations, as she used to be. She no longer plays with her two companions (cynocephali), and remains indifferent, or pays but transient attention when they come near. She is not jealous when they are petted. Threatened, she evinces fear, but does not show any of the signs of self-defence and revolt she used to evince with so much energy. A feeling of indefinite and baseless terror is manifested, as soon as anyone goes near her, or her automatic agitation is suspended by a sudden call, or by any other new impression.

The movements of the left side are more precise, but not quite normal, though she uses it to take food, or steady herself in saluting. The vision of the left eye has, to a great extent, returned; but, though it is difficult to determine the degree with precision, it is certain that the internal portion of the visual field is more acute.

She continued much in the same state till January 21, 1893; and still has some tendency to rotation. She does not use much the left arm. She walks with agility and shows no trace of paralysis, using for that purpose the left arm. Sensibility intact in every sphere. She is killed with chloroform.

The left hemisphere looks larger than the right. The middle and inferior frontal convolutions are destroyed as far as their insertion into the anterior Rolandic gyrus, which is almost intact. (fig. 1). The superior frontal has hardly been touched as far as its anterior extremity. In the right hemisphere, the superior frontal has been destroyed up to its root, excepting a small portion communicating with the rest of the brain by a narrow bridge (2 mm. in width). The second, or mid-frontal, is more extensively destroyed; of the third frontal, only the anterior and central portions are less destroyed. (fig. 2).

(*c*) A young female cynocephalus, had been an inmate for about two months, whilst she was well treated and had become domesticated. She first menstruated during this period, and is much attached to one of the servants. She is docile, obedient, inquisitive and lively.

On *August* 2, 1893, I freely expose the right anterior hemisphere. Faradic excitation provokes no contraction in front of the points *a* and *b*. In *a*, movements of the trunk and pelvis were observed; in *b*, movements upwards and forwards of the ear, of dilatation of the palpebral slit, with elevation of the upper lid, and of dilatation of the pupil (chiefly on the same side). Repeated observations constantly gave the same results. Slight increase of current strength, or displacement of the electrodes 1 or 2 mm. backwards, movements of the hand (extension of the fingers chiefly) ensued. By a deep incision with a sharp curved blade I remove all the portion, one millimeter, in front of the points *a* and *b*. About six grammes of cerebral substance are removed. The wound was cleaned, under strict antiseptic precautions, and closed in the usual manner.

August 3.—Sleepy the whole day. Sits, does not lie down. No deviation of head and eyes. Uses the hands, preferably the right; walks about the cage normally. Drinks some milk.

August 4.—Same condition; is sick once.

August 7.—Has recovered from the operation; the wound has healed by first intention.

This animal is to all intents and purposes in a normal condition, save in two particulars, which only a searching examination can reveal. She is subject to fear, and has left external hemianopsia. She walks well; no rotary movements. Turns the head about with no difficulty to either side; is as rapid and agile as before. She perceives contact equally all over, including head and neck. Sudden prickings with a fine needle are followed with the most lively reactions.

If vision is prevented by means of sub-palpebral membranes of india-rubber (after cocainisation), the animal is found to have normal muscular sense. A peach brought near her hands is seized, bitten, skinned, and the pieces carried to the mouth with either hand, and with perfectly accurate movements. The sound of a diapason is heard, and causes fear, at the same distance of either ear. Smell and taste were not systematically tested.

From the day of the operation a defect of sight on the opposite side was noted. She was held by an attendant, whilst another kept her attention fixed on a point in front of her. From behind I bring a piece of peach to 10 or 15 cm. from the lateral or upper part of the visual field. When the object, travelling from outside towards the external angle of the right eye arrives in the field, it is immediately perceived, and sometimes rapidly seized and eaten. A similar piece brought towards the left eye has to be brought much farther forwards, almost as far as the median vertical plane, before it is perceived.

August 12.—On repeating the examination, the difference between the two eyes is found to be less precise and definite. She perceives something, but recognises it only when it reaches the left median vertical plane, the vision of the right eye being normal as before.

September 11.—She is still alert, agile, observant; she unties the string which closes the door, and pulls back the bolt and comes out. She is fond of tobacco. The end of a lighted cigar is thrown to her; she picks it up with left hand, burning herself; she speedily lets it go and licks her fingers; soon after she picks it up cautiously by the wet end, examines it, unrolls the leaves, which she chews one by one.

Up to January, 1894, no changes were observed, except that she was more irritable.

January 31, 1894.—Under morphia and chloroform, the anterior left hemisphere is exposed. The ascending frontal and the posterior portion of the first frontal convolution remain covered. Faradic excitation provokes contractions in the third frontal, where in front of the face centre (contractions in the inferior half of the face) a vertical rectangular area is determined, extending upwards to the second convolution, in which excitations arouse feeble opening of the lids, a slight raising of the eyebrow, and dilatation of the pupils, especially on the opposite side. At the origin of the first frontal, and a little in front, excitations provoke rotation of the head to the opposite side. No other movements could be elicited. (Repeated excitations of

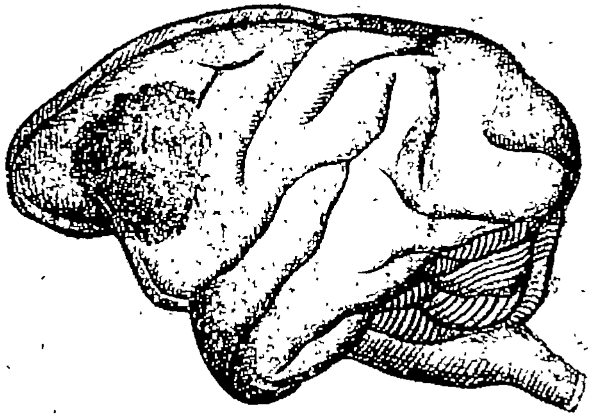

FIG. 1.

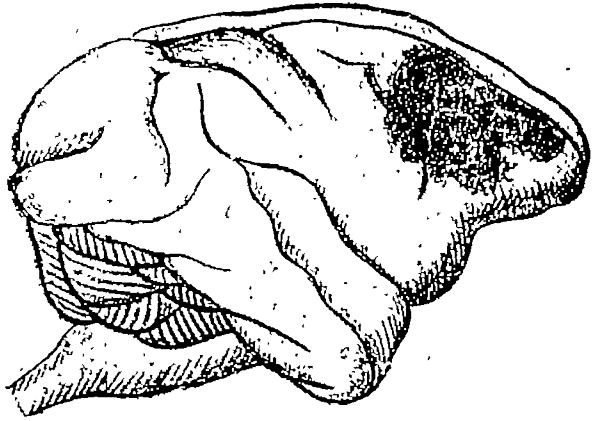

FIG. 2.

The Fissure of Rolando should be prolonged further down.

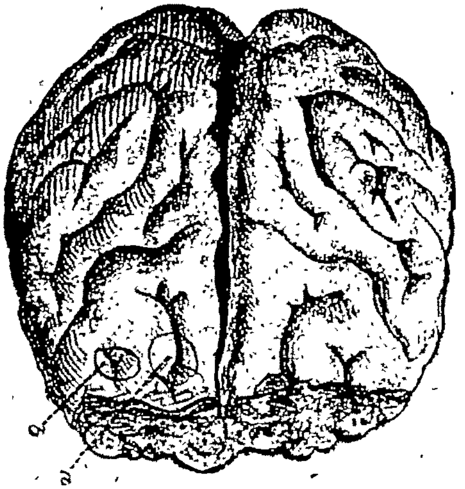

FIG. 3.

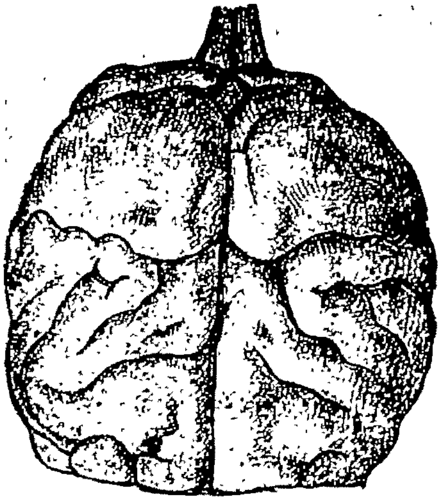

FIG. 4.

the pupillary centre produce persistent opening of the opposite lids, lasting about ten minutes.)

The incision is made immediately in front of the face centre, and behind that of the dilator iridis, and all the anterior portion of the brain (excepting the posterior third of the first frontal) is removed. Little hæmorrhage; antiseptic dressings. The animal remains for some hours in a somnolent condition; but opens the eyes when called. No vomiting nor fever.

February 2.—Remains in the cage with head bent, eyes half open, sleepy but sitting. When called she raises her head, slowly opens her eyes, looks about, and relapses into the same condition. When touched she wakes up, sometimes with a start or a cry, but does not try to escape nor looks alarmed. When let loose in a room, she walks about with measured steps. No deviation of the trunk nor paralysis, except in the right inferior half of the face.

February 9 and 14.—The notes of these two days may be taken together. Facial paresis not so marked, though evident. On walking, the lower part of the body looks arched. She can turn to either side, bend on herself, runs, without any trace of paralysis. Both sides of the body seem equal. Only she has lost her normal elasticity and suppleness; she is duller, heavier, more relaxed, even when urged on. She uses either hand, but preferably the left; there is still a certain precision but less decisiveness and security in the use of the right. No neck paralysis; all movements of the head, in every direction, are possible. She eats well, with avidity, swallows and utters cries as before. Tactile sensation preserved; she reacts to sudden contact of every portion of the body. Perhaps she feels less when the left ear is touched or pricked, as she does not move the part, or starts when it is done to the other ear. She scratches herself, and looks for parasites on both sides and with both hands. The muscular sense appears thus to be intact; no ataxia on walking, or performing delicate movements has been noticed.

As to sight, closure of the left eye does not interfere with walking, or running, all obstacles being accurately avoided. If, however, whilst she is at rest, a piece of sugar, tied to a string, is brought from the outside inwards, into the right visual field, it is perceived only when it is in a line with the visual axis. She then rapidly seizes it with the left hand and sometimes with the right, though it is less mobile. This experiment gave constant results. On the other hand, when the right eye is closed, the sugar is perceived and the movement for seizing it made the instant it is brought into the left field of vision. No marked difference between the upwards and downwards extent of the two fields of vision; the left internal field could not be examined on account of the fatigue.

Intellect.—Attitudes stupid, loss of physiognomical expression; apparently aimless walking. She runs incoherently from one end of the room to the other; when she reaches the wall she stops, returns to the opposite angle. Noticing a defect on the wall, where bits of plaster have become detached, she picks them up, chews and swallows them; the same with bits of filth on the ground. Pieces of plaster offered to her are taken and eaten as if they were sugar. It is not because she does not distinguish between these substances; for she takes longer to chew the plaster and seems at times to hesitate whether to go on or not; but she cannot resist the idea of sugar, which the appearance of the plaster has evoked. In all her actions there seems to be a defect of perception, which seems to be reduced to a more elementary level and lack some of the factors necessary to the formation of a more complete judgment. Her psychical life seems to be reduced to the existence of actual sensations.

Reflex excitability is exaggerated; every noise startles her, and she has lost her courage. On a threat she runs away, with incontinence of urine, or huddles herself up in a corner uttering lamentable cries. She displays no resource in her flight or attempts to hide herself, does not turn on the assailant; experience does not profit her, and as often as a sham attack is made upon her, she goes through the same actions as if she did not remember what had happened just before.

Formerly she used to cling affectionately to the legs of the attendant, caressed him, and nestled on him with the expression of the utmost pleasure and love. Now all seems indifferent; she stops at nothing; looks at everything but without fixing herself to anything. Her senses are excited but not attracted. She remains in this condition for months.

On *May* 4 she was chloroformed. The extent of the lesion is shown in fig. 3. No damage to the olfactory apparatus.

(*d*) Cynocephalus, female, good size, agile and cheerful. On close examination no defect of the senses, organs of motion, or of any other kind was discovered. She is inquisitive, cunning, acute, attached to her keepers. During the menstrual periods, which are regular, she is more excited and mobile than usual.

May 20, 1893.—Under morphia and chloroform she was trephined over both frontal lobes, one cm. in front of the Rolandic sulci. Little hæmorrhage. The brain was exposed and two lines marked symmetrically on each side, passing in front of the roots of the frontal convolutions nearly corresponding to the præfrontal sulcus. Faradic excitations in front of these lines provokes no contractions, either in deep chloroformisation, or in mere morphinic somnolence. The whole of the brain substance in front of the lines was removed, and the wound dressed, under strict antiseptic precautions, in the usual manner.

May 21.—Stupor; marked reflexes to the least noise. She dips her snout in milk, hardly drinking any. Internal temperature 38·6° C. The next day the symptoms remained much the same. Appetite better. She was kept in her bed, and a clyster given.

May 27.—More awake, but the whole head is œdematous. She can, however, be examined. Sensation to pain and touch exaggerated, especially on the left side. The least contact, even to the tips of the hairs, is followed by reflex movements of the whole body, and turning of the head and eyes to the excited side. The slightest pinch, or pin-prick, elicits loud cries. A diapason suddenly set vibrating on either side astonishes her; she moves the head as if to avoid the strange noise. If the hand or any other object is brought towards the eyes, from whatever point in the visual field, she blinks and withdraws the head in fear. If a cherry, of which she is fond, is thrown, she fixes her eyes on it. If, suspended to a string, it is brought from above, or from the outside, into the visual field, she recognises it, and tries to take it, her movements being appropriate and co-ordinated with the mouth or the left hand. She walks badly, swaying, with head and body bent; the left leg drags, and the right arm little used. She hardly ever leaves her bed, nor takes any interest in things; but starts, emitting cries as if afraid or angry, to any noise, contact, or whenever she is attended to.

June 3.—The swelling has gone down. There is a local suppuration, and a superficial abscess has to be opened, cleaned and dressed. Her condition remains much the same; but she eats more, resists better, and walks, though with some uncertainty. The right leg is much paralysed.

June 7.—Suppuration has stopped. Wound granulates, and is still dressed antiseptically. No fever since the operation. The animal astonishes us all by its novel behaviour. She remains in the position given to her. No curiosity nor interest in what goes on. She spends hours sitting with head bent, as in a lump, often asleep. If a noise is made she shakes herself up, often with a cry, raises the head, looks about, and relapses in her previous condition. The same occurs three or four times together if the same noise is repeated, until she is used to it. The only thing that excites her interest and makes her move is the sight of some favourite fruit dropped at a short distance. She then rises and runs to seize it. But if it is dropped further off she does not move; and it is difficult to say if it is because she does not see it, or through apathy. She occasionally scratches herself, but no longer hunts for fleas with her former rapidity and precision of movement. She resists if pushed away, and if she can cling to the place she occupies, she sticks to it. If pushed along by the nape of the neck she walks along semi-automatically, preferably along a wall, and stops in a corner, where she remains for a long time. When called aloud she looks up, but soon the head falls again, with frequent escape of saliva. She is very dirty, and does not move when passing her urine and fæces in which she sits. Placed with another menstruating female cynocephalus she stopped automatically, quite insensible to the erotic and lascivious manifestations of which she was the object, with no sign of sexual or racial interest whatever. And yet a noise startles and frightens her; a threat with a stick terrorises her, whilst her companion evidently recognises the sham nature of the demonstration. Fruits, sugar, and the like excite her more, but yet she lacks discrimination. A cherry into which a solution of quinine has been introduced is given to each monkey. The normal animal, after a bite or two, spits it out with a disappointed and irritated look, cleans her tongue with her hand, and crushes the deceptive fruit on the ground until no trace of it is left. The other chews, but stops after a while, salivating abundantly, and after some hesitation ends in swallowing it. After giving to each some good fruit, I give to each a cherry, dipped in the bitter solution. The first animal, as soon as it has tasted it, spits it out in anger; but soon after, seeing that the fruit is whole, picks it up again, examines it attentively, opens it carefully, eats the flesh and throws away the skin. The second goes through the same process of chewing, stopping, and finally swallowing the fruit.

A piece of sugar was given to the normal monkey; next a piece of plaster of the same shape and size and appearance. The animal picks it up, looks at it, sniffs it, and throws it away without tasting it. After giving it some natural cherries, I presented to it one made of wax. The monkey likewise scrutinizes it thoroughly, drops it as if to test the sound, tries to rub it, and finally leaves it alone. The operated animal in both experiments displays a striking want of discrimination; it eats and swallows the plaster as it had done the sugar, and without any examination tries to chew a piece of wax, though not of the same shape as a cherry, giving us trouble when we tried to remove it from its mouth.

July 15.—This monkey has been kept under observation with reference to the changes in its temper, character, instincts and intellect. Her behaviour is altered, her physiognomy stupid, less mobile; the expression of the eyes is as if uncertain and cruel, devoid of any flashes of intelligence, curiosity, or sociability Shows terror, even by shrieks and gnashing of teeth, when threatened or hurt, but never reacts aggressively. She is in a state of unrest; when placed in a large closed room, it walks aimlessly round it, always in the same direction, without stopping near any object or person. Any action done with apparent purpose remains incomplete, unfinished; if she runs towards a door she stops near it, goes back, runs to the door again, and so on several times.

She shows no affection nor gratefulness to people whom she previously loaded with caresses, as these animals are wont to do; she does not seem to know them, though they are ever ready to attend to her wants. Whenever approached for a caress she shows fear. When the attendant brings her food or fruit, she comes near and violently seizes the object of her avidity. None of her former friends can now caress her any longer. She is unsociable with the other monkeys, does not play; cannot overcome the least difficulties in her way by new adaptations, nor learn anything new, nor recover what she has forgotten. She picks up and takes to her mouth whatever she comes across. She is, however, somewhat cleaner.

The sexual instinct seems to be present; but the periods are less regular and abundant. She occasionally manifests impulses of cruelty quite foreign to her kind. One day whilst she was menstruating, another female cynocephalus came near her. She at once manifested her desire; but finding the other unable to satisfy it, she attacked her companion so furiously that she would have killed it if the keeper, armed with a stick, had not at once interfered.

A new psychomotor "tic" has recently been observed; every ten or fifteen minutes whilst walking, she stops suddenly, and placing the foot on the nape of the neck, bites her right buttock (sometimes the left also). Occasionally she proceeds to bite also one heel, then the other, after which she quietly resumes her walk. No apparent local cause can be discovered for these actions, which are always identical, almost rhythmical. It is possible that at the outset some abnormal sensation has given a start to them, and that they afterwards were persevered in, governed by an uncontrollable automatism.

August 15.—Psychical state unchanged. Her movements have a stereotyped character, but are faultless. New adaptations remain impossible. She walks about, sits, looks for parasites all over the body, remains indifferent to everything but to the sight of her food. She often puts the parasites into her mouth, which she used not to do before. She also eats dry leaves or flowers. She no longer expresses her desire, at the time of menstruation, as she used to, by appropriate sounds and gestures.

No sensory defects are noticeable, on the closest investigation. But discrimination, and the higher co-ordinations of sensory factors on which the more complex psychical manifestations depend, and which are necessary to the preservation of the individual and the species, are reduced to mere rudiments. She now differentiates between a sugar plum and a piece of plaster. She eats the first with avidity; the second she breaks in pieces with her teeth, but spits them out on the floor. But then she takes one up to her mouth, throws it down again, and so on several times, at last swallowing it with avidity; she next takes a second, and so continues until the whole number has been disposed of. The whiteness of these fragments and their resemblance to those of a sugar plum have prevailed over her sense of taste. Her rudimentary inhibitory power has been overcome by the visual image; the other sensory factors did not intervene in the act of identification, in the judgment of analogy. She has crude sensations, but no co-ordination of simple presentations, into representations of higher complexity. Another monkey takes the plaster plum, examines and smells it, and either rejects it at once, or, if deluded by the visual resemblance into chewing it, spits it out and ignores the fragments; its comparative judgment and memory are certain, and spare it the struggle which, in the abnormal subject, was followed by a victory of the more elementary sensation, and by an irrational impulsive action.

August 23.—No change. The animal is chloroformed, and the right hemisphere exposed. Electrical examination of the cortex proved the existence of all the normal motor centres of the limbs and face. The brain is shown in fig. 4.

Conclusions.

Granted the objective accuracy of the facts just related, it will not be difficult to give a plausible explanation of the phenomena observed after ablation of the frontal lobes. But even if such a hypothesis be not universally accepted, the value of the observed facts will not be thereby diminished.

It is useless to discuss at great length Munk's views on this subject. Paralysis of the trunk-muscles does not always occur, especially when the incision falls accurately on the frontal lobes; and is in any case but temporary. Moreover, what connection can there be between such a paralysis and alterations of temper? It is strange that the Berlin physiologist should assign to such a function a centre so vast as to constitute a true province, far wider than that of the arm with its prodigious scope for movements of the highest complexity. And it is surprising to find the paralysis disappear some weeks or months after the experiment performed to disprove the narrow limits ascribed to the cortical area of a so much higher function.

We readily understand Munk, when with Meynert, Ferrier, and Goltz he refuses to admit a centre for a so-called intelligence, as he expresses himself. But it is astonishing that such an acute observer should have overlooked so many psychical symptoms, whilst attributing to the extensive frontal lobes, a function shown by Schäfer and Horsley to be localised in a much more modest region (internal aspect of marginal gyrus).

No scientific man at the present time dreams of galvanising into life the dead body of Gall's organology. Every one of the several sensori-motor areas of the cortex combines with the others in the formation of the psychical personality, whatever its characteristics may be; nay, the whole of the nervous system takes part in it. Nobody will contest Munk's thesis in this particular; but these remain to account for all the phenomena observed after the destruction of the frontal lobes, and which are not explained by the occurrence of a temporary trunk-muscle paralysis.

Neither does Ferrier's hypothesis satisfy us. Our facts point to more than a simple defect of attention correlated as much with a paresis of the ocular and cervical muscles as with a non-existing cause. Dogs and monkeys, a few weeks after the operation move their heads and eyes in every direction; and there remains nothing of this paralysis to explain the deep psychical changes observed by us. Such disorders, therefore, depend upon the absence of the frontal lobes, not as the motor centres of the head and neck, but as the anatomical substratum of special functions on which the formation and manifestation of psychical personality depend. The frontal lobes are not centres of inhibition. We speak too assuredly and too lightly of attention, and of inhibition, and one may feel surprised to find Ferrier identifying certain muscular co-ordinations with the very essence of attention, being thus led to spread over the whole frontal area the motor centres of the head and neck, in order to assign to it the functions of cortical seat of attention.

But we may ask ourselves whether there exists really any centre the function of which is to inhibit, hence whether there exists a faculty of attention. I reply distinctly, No.

Without attempting to go fully into the theories concerning inhibition and its centres, it will be granted that inhibition depends upon a general psycho-physiological process, and that every part of the nervous system becomes, under different circumstances, either an inhibitory or an inhibited centre. I upheld this view in 1885 in an essay on the act of walking, in which I modified in a more physiological sense the theory of Lauder Brunton, who explained inhibition by the law of interference. Taken in its strict sense, interference bears no likeness to inhibition: we must rather speak of an afflux of nerve-waves into a region excited by some definite stimulus or psychical representation, which afflux weakens the aptitude of other regions to fulfil its function. This mechanism can be illustrated by the action of any of the various centres, auditory (as when we are absorbed in listening to a musical performance), visual (as in presence of a great picture or object of scientific interest), or motor (as in the case of a juggler or acrobat engaged in wonderful muscular display). Each respective cortical area becomes in its turn an inhibiting centre for the others. Physiology and psychology swarm with facts showing the error of those who assume a special cortical centre for inhibition.

We know how, when the mind is busy with a scientific problem, one becomes, as it were, deaf and blind. But if the auditory or visual centres are then strongly excited by a voice or an image, and a perception aroused, the flow of ideas is arrested or weakened as long as the psychical disturbance persists in another portion of the brain.

Thought, even when at work in the domain of pure conceptions, has in most men an inhibitory power. This is due to the fact that abstractions are resultants of very numerous psychical elementary factors, and determine the same dynamo-molecular processes in the innumerable nerve elements which co-operate in the formation of each concept. Hence the frontal lobes are no more centres of inhibition or attention than they are motor centres for the trunk, or head and neck muscles.

Overlooking the possible contention of some objectors, that the serious mental disturbances observed in the mutilated animals, depended on interference with the senses of taste and smell, my hypothesis is that the frontal lobes are the seat of co-ordination and fusion of the incoming and outgoing products of the several sensory and motor areas of the cortex. As the nervous waves from peripheral organs of reception (retinal rods, tactile organs, &c.) are transmitted from neurons of the first order to neurons of the second (mesocephalon, thalamus), and from these again to neurons of the third order (cortex), thus we may suppose that from the last-mentioned, nerve impulses travel to the frontal neurons of the highest order. The frontal lobes would thus sum up into series the products of the sensori-motor regions, as well as the emotive states which accompany all the perceptions, the fusion of which constitutes what has been called the *psychical tone* of the individual. Removal of the frontal lobes does not so much interfere with the perceptions taken singly, as it does disaggregate the personality, and incapacitate for serialising and synthesizing groups of representations. The actual impressions, which serve to revive these groups, thus succeed one another disconnectedly under the influence of fortuitous external stimuli, and disappear without giving rise to associational processes in varied and recurrent succession. With the organ for the physiological fusion which forms the basis of association, disappear also the physical conditions underlying reminiscence, judgment, and discrimination, as is well shown in mutilated animals. Their agitation and motor incoherence depend upon the reflection of nerve impulses set up by stimuli through small sensori-motor arcs, without the intervention of the previously accumulated psychical co-efficients.

Fear is an immediate result of psychical disaggregation, from defective sense of personality, and unbalanced perception and judgment. As the oyster closes its valves on the approach of a cloud, thus the operated monkey trembles at the simulated hostility of its keeper, ceases to read on his face the smile of kindness, does not improve by past experience, nor perceive the means of escape at hand. Courage rests upon the treble basis of self-conscious force, rapid perception of the enemy's powers for offence or defence in relation to one's own, and the influence of certain feelings; our animals show an absence of all these characteristics. (We must not mistake for courage the impulsivity of certain animals; or men, insane or epileptic.) On the one hand, their effective nature, friendliness and sociability is impaired; on the other, their avidity becomes reckless and insatiable. From cleanly they become dirty; and their sexual functions are perturbed; the females menstruate irregularly; the males become, if not incompetent, at least incapable. All these symptoms depend not upon motor deficiences in the head, neck, or trunk muscles, but upon a dissolution of the psychical personality.

But even should a more acceptable hypothesis to explain the facts observed be hereafter framed, I feel at any rate certain of the accuracy of the observations themselves.

DEFECTIVE DEVELOPMENT OF THE CEREBELLUM IN A PUPPY.

BY J. S. RISIEN RUSSELL, M.D., M.R.C.P.

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(From the Pathological Laboratory of University College, London.)

CONSIDERABLE interest is attached to the condition of the cerebellum in this puppy, both on its own merits, as illustrating some important points in the pathology of cerebellar disease, and also when considered in conjunction with a case of defective development of the cerebellum in a young cat, which I published in a recent number of this journal.[1]

I am indebted to Professor Victor Horsley for placing the animal at my disposal; and my best thanks are also due to him for allowing me to make the necessary examination of its central nervous system in the Pathological Laboratory of University College.

The animal in question is one of a litter of puppies all similarly affected, though born of healthy parents, both of whom were prize dogs. Unfortunately this was the only puppy obtained for examination, and came into Professor Horsley's possession through the kindness of Mr. Sewell, F.R.C.V.S.

During life it presented in a marked degree the clinical picture which we are in the habit of associating with some defect or disease of the cerebellum. Inco-ordination[1] was so great that in attempting to walk the animal fell, sometimes in one direction, sometimes in another, there being no one direction in which it was inclined to fall more frequently than in any other. When standing, and even when sitting there was almost constant oscillation of the head and trunk. The most careful examination failed to detect any clinical symptom that might be taken as an indication that one lateral half of the cerebellum was more affected than the other, and the correctness of this was borne out by the subsequent *post-mortem* examination of the central nervous system.

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[1] BRAIN, part i., 1895.

The animal was killed by an overdose of chloroform, and the central nervous system placed immediately in Müller's fluid for preservation. Sections were subsequently cut in celloidin, stained by Schäfer's modification of Pal's method, picro-carmine, aniline blue-black, hæmatoxylin, eosin, &c., and examined microscopically.

In the present description of the results of the macroscopic and microscopic examination of the central nervous system, the condition of the cerebellum will be dealt with first, those structures in other parts of the central nervous system which are known to be intimately connected with the cerebellum will be next considered, and finally the deductions which seem warranted will be submitted.

The Cerebellum.

On macroscopic examination of the organ it was evident that it was reduced in size so as to be about three-fourths the size of that of a normal puppy; but it was quite symmetrical, and the general contour of the organ was well preserved. Its convolutions and sulci appeared to be as much differentiated as in the normal cerebellum, and but for its general reduction in size, and the fact that the breadth of the convolutions was diminished, it might have passed as such.

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[1] I think it well to mention in this connection that I am familiar with the fact that the co-ordination of a normal puppy is not of the best, and that I am in a position to state that the inco-ordination presented by this animal was far in excess of anything I have ever seen in the normal puppy, and left no doubt in the minds of all of us who saw it that some defect of the cerebellum must be the cause of this.

Sections through the organ showed the proportion of grey and white matter to be on the whole not much altered in some places, the general normal arrangement being also preserved, but the folia looking somewhat skrunken. In other parts the cortex of the cerebellum was distinctly of less depth than it ought to have been, while in other places, and these were fewer, it appeared to be of greater depth than the normal.

The microscopic examination of the organ confirmed the general impression obtained from the macroscopic examination, inasmuch as in parts the proportion of grey cerebellar cortex to white matter was, as nearly as could be judged, normal, while at other parts the cortex was distinctly reduced in thickness, and in a few places it was of greater thickness than it ought to have been. Further examination revealed the important fact that the different layers of the cortex of the cerebellum were variously altered in their relation to each other. Thus in one part the molecular layer was three or more times the proper depth as compared with the normal or with the granular layer at this part (see fig. 1); while in another place the molecular layer was about one third or a half the depth of the normal, or as compared with what it ought to have been to be in proportion with the granular layer at the same point (see fig. 2). In other parts again the depth of the molecular layer, as compared with the normal or as compared with the granular layer, was as nearly as possible normal. In some parts of the cortex the molecular layer was of much greater depth than normal at one point, and greatly reduced in thickness at another, the transition from the one to the other being quite abrupt. A good idea of the extremes which were met with in places, side by side, may be obtained by comparing fig. 1, in which the molecular layer is greatly increased in depth, with fig. 2, in which the same layer is greatly reduced in depth. Some idea of the abruptness of the transition from the one condition to the other may be obtained from an examination of fig. 2, as in the extreme left of the figure, beyond the cluster of cells of Purkinje, the molecular layer may be seen to be about four times the depth of that part of it above the cluster of cells and throughout the rest of the upper margin of the specimen.[1]

All that has been said with regard to the variations in the depth of the molecular layer in different parts, applies also to the granular layer, which presented all variations from the normal, being increased in thickness in some places, reduced in others, and preserving a fairly normal thickness in others.

Striking as were the appearances presented by the molecular and granular layers of the cortex of the cerebellum, the condition and arrangement of the cells of Purkinje were of still greater interest. In large parts of the cerebellum these cells were completely absent (see fig. 3), in other parts one or two were to be seen here and there, while in a few places irregular groups or clusters of these cells were to be seen, the outer margin of the cluster being at the junction of the molecular and granular layers, while the cells invaded the granular layer to a considerable extent (see figs. 1 and 2). One or other of the conditions already described was that met with as a rule, but in some places there was a greater tendency to regular arrangement of the cells of Purkinje in a single line in their normal position between the molecular and granular layers: but even here the distances between the cells were very irregular and often much greater than normal (see fig. 4).

Not only the arrangement of the cells of Purkinje was altered, they also presented all degrees of variation in shape and size. While in places these cells varied no more in shape and size than in the normal cerebellum, in other parts which formed the greater proportion, the cells presented the most varied shapes and sizes, many being much below the normal size.

One other point to be noted is the fact that the greatest paucity of the cells of Purkinje was met with in the lateral lobes of the cerebellum. In the middle lobe, on the other hand, there was more regularity of arrangement, and a greater richness in these cells.

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[1] The photograph does not show this point as well as it might have done had a little more of the molecular layer to the left of the cluster of cells of Purkinje been included in the field.

Corpus Dentatum.

This structure was absolutely intact on both sides, as seen in stained specimens both on macroscopic and microscopic examination. Its cells were normally arranged and individually presented no abnormal appearances.

Cerebellar Peduncles.

These were all reduced in size in proportion to the general reduction in the size of the cerebellum, but none of them showed any evidence of sclerosis or recent degeneration.

The Medulla Oblongata.

Inferior Olives.—The structures to which attention was primarily directed in this part of the central nervous system was naturally the inferior olives, owing to the intimate association known to exist between these structures and the cerebellum. They were of perfectly normal macroscopic appearance, and under the microscope no evidence of any abnormality could be detected, either in the characters of their component cells, or in their arrangement (see fig. 5).

The Restiform Bodies.—The region occupied by these peduncles of the cerebellum on both sides showed no evident defect in the way of absence, sclerosis, or more recent degeneration. Nor was any sclerosis or degeneration to be traced to either inferior olive.

The Spinal Cord.

Nothing abnormal could be detected in this structure. None of the tracts known, or supposed to be connected with the cerebellum, showed any evident reduction in size, sclerosis or recent degeneration. The grey matter was everywhere normal in appearance.

The Red Nucleus.

Inasmuch as experimental evidence has proved that so many of the fibres which leave the cerebellum by its superior peduncles, and which degenerate after ablation of some part of the organ, terminate in the red nucleus, careful search was made for any sign of defect in this structure, with negative result.

The Optic Thalamus.

Like the red nucleus, the optic thalamus is the recipient of a large number of the fibres which leave the cerebellum by its superior peduncles, as has been demonstrated by the degeneration method after ablation of parts of the cerebellum, so that special attention was paid to the appearances presented by this structure, but nothing abnormal was found.

The Cerebral Cortex.

To the unaided eye the appearances seemed quite normal; there was no detectable atrophy or shrinking of the convolutions, especial attention being paid to the condition of those of the sigmoid and adjacent gyri.

Auditory Nerve and Labyrinth.

No disease or defect was found in connection with these structures to account for the loss of power of equilibration which was so prominent a symptom during life.

Concluding Remarks.

The appearances met with in the cerebellum in this animal made it clear that the defects were of congenital origin, as indeed must be clear from the description of them contained in this paper. There was nothing in the appearances to suggest the existence of any disease of a progressive character, it is therefore interesting to find so much disturbance of equilibration, for animals deprived of even the half of the cerebellum, experimentally, regain their powers of equilibration in a remarkably short time. Possibly the absence of compensation in this puppy was due to the fact that those structures by which compensation is ordinarily brought about, after lesions of the cerebellum, were themselves in an imperfect state of development at so early an age.

The next point which calls for comment is the fact that a defect limited to the cortex of the cerebellum, and affecting in such a great measure the cells of Purkinje, was attended during the animal's life, with symptoms which led those who saw the puppy to suspect some defect of the cerebellum.[1]

This in itself is a fact of no small importance; but as important is the fact that in spite of these changes in the cortex of the cerebellum, no evidence of any defect in the inferior olivary bodies could be detected. That no changes could be detected in the inferior olives is not surprising when we remember that the corpus dentatum was, as far as could be judged, absolutely intact on both sides. The intimate relationship which exists between the corpus dentatum and inferior olive has been too abundantly proved by experimental and other evidence to need further emphasis here; but it is gratifying to find confirmatory evidence of this fact in the animal under consideration.

The contrast between the condition of things in this puppy and that which existed in the cat,[2] to which I have already alluded, is so striking, that I cannot refrain from calling attention to it. Whereas in this puppy the defect was limited to the cortex of the cerebellum, and with the corpus dentatum intact on both sides, the inferior olives were also both intact, in the cat the defect (a unilateral one) involved the corpus dentatum to such an extent on the affected side that its only representatives were a few scattered cells in the region where it should have been found, and in conjunction with this condition the opposite inferior olive was extremely diminutive, contrasting markedly with its fellow. More important evidence, short of experimental, of the all-important relationship which exists between the corpus dentatum and inferior olive, as contrasted with any relationship between the cells of Purkinje and the inferior olive, could not easily be obtained.

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[1] *Cf.* Herringham and Andrewes, *St. Bartholomew's Hospital Reports*, vol. xxiv.

[2] *Loc. cit.*

DESCRIPTION OF PLATE.

FIG. 1.—Showing increased depth of the molecular layer of the cerebellum and a large cluster of cells of Purkinje to the right of the figure and an absence of these cells to the left of it.

FIG. 2.—Marked diminution in the depth of the molecular layer throughout the greater part of the figure, but greatly increased depth of it to the extreme left of it. Scarcity of cells of Purkinje except at the point of transition from the increased depth to the diminished depth of the molecular layer, where there is a group of them.

FIG. 3.—Complete absence of the cells of Purkinje, and diminution in the depth of the molecular layer

FIG. 4.—Cells of Purkinje arranged in single line between the molecular and granular layers, but at very irregular distances from each other.

FIG. 5.—The inferior olivary bodies presenting no abnormal appearances.

Fig. 1

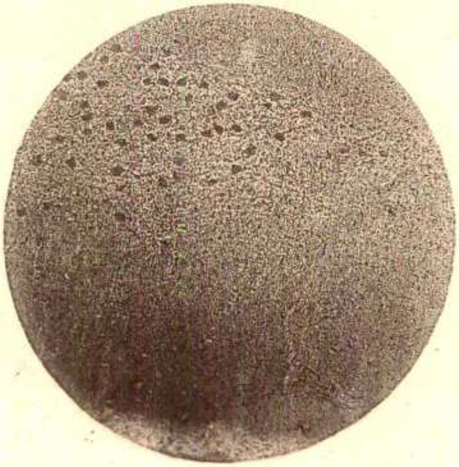


Fig. 2.

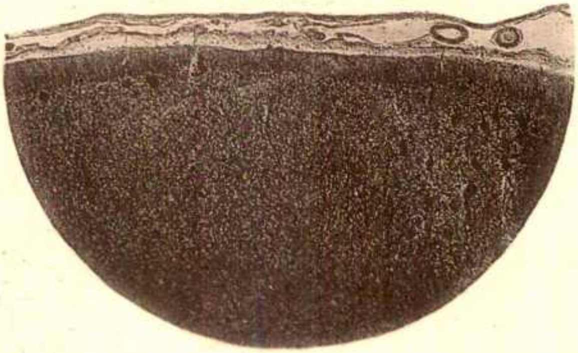


Fig. 3.

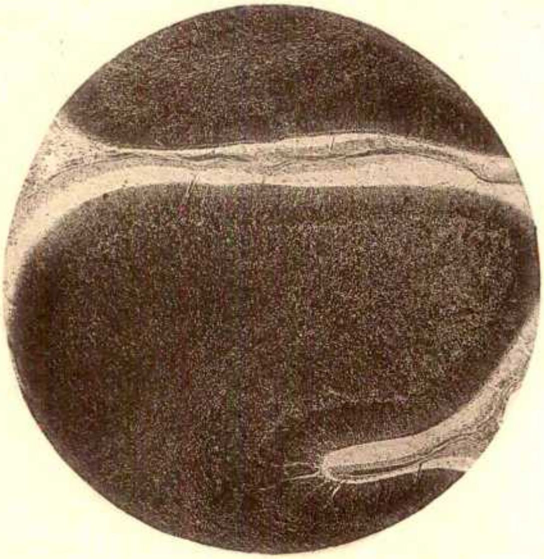


Fig. 4.

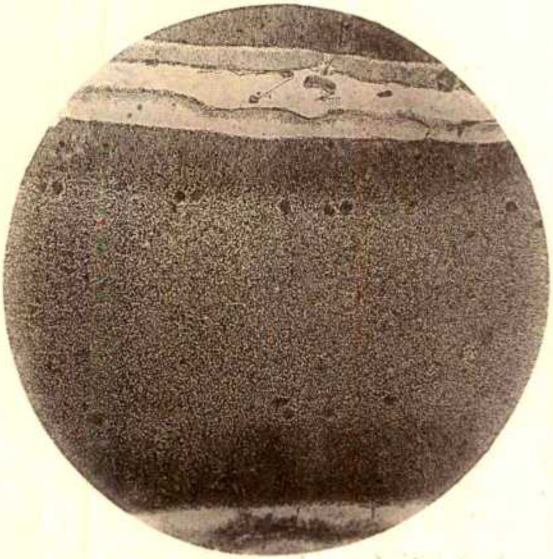
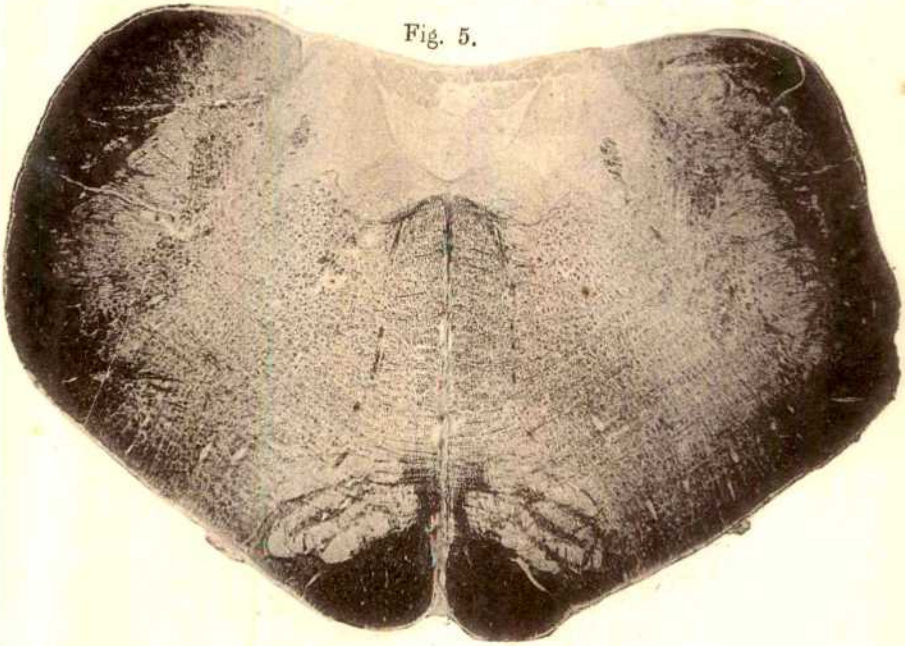


Fig. 5.



Danielsson & Co., London. Collotype.

DR. RISIEN RUSSELL ON THE DEFECTIVE DEVELOPMENT
OF THE CEREBELLUM.

ON TUMOURS OF THE CORPUS CALLOSUM, WITH AN ACCOUNT OF A CASE.

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THE paper is based on the following case:—

Mrs. E. W., aged 24, married two and a half years, but never pregnant, came on September 8, 1893, as an out-patient to the General Hospital, Nottingham, for general nervousness and fits.

It appeared that her family were of an excitable, neurotic disposition, and that since marriage she and her husband had led a restless, pleasure-seeking life, and indulging rather freely in alcohol, although not so as to cause intoxication. There was no history of syphilis, congenital or acquired.

She was said never to have had any fits till two years ago, when the first one occurred after a mental shock. The fits have occurred as often as every other day, but for the last few months only once a week. The fit is preceded by a few moments of general excitement, then occurs a peculiar sensation in the temples; loss of consciousness and spasms following. These consist successively of twitching of the left hand and arm, left leg, left side of face, and deviation of the head and eyes to the left. The tongue has not been bitten, but urine may escape involuntarily.

The fits only last a few minutes, and are not followed by hysteroid attacks. The patient's natural excitability has much increased during the last year, and headaches, to which she has all her life been liable, have become more frequent and severe. The pain is most intense in the temples and the nape of the neck. For the last six months the eyes have become prominent and staring, but the neck has not enlarged, and she has not complained of palpitation or sweats. The pulse rate on this date = 120.

Under treatment with moderate doses of bromide the fits stopped entirely, but the mental excitability increased so that her family could hardly manage her.

On *October* 18 she was admitted as an in-patient, being then in a semi-maniacal condition. Her violence was at first such as only to be controlled by an injection of hyoscyamine, but began to lessen on the second day after admission.

State on October 20.—The patient is still restless and excitable and rambles in her talk, having some ideas of persecution. She says her headache has gone. The facial expression is anxious and frightened; there is marked exophthalmos, and the voice is somewhat tremulous. The temperature is normal; the pulse is very small and soft = 120; thyroid gland not enlarged. The heart and lungs are normal to examination. Though the tongue is coated, the appetite is good, and there is no sickness or other disturbance of the digestive system. The bowels are rather constipated. Generative organs normal. There is no paralysis, spasm, or alteration of general sensation, and the special senses appear normal.

There are slight general tremors of limbs on movement, and the gait is a little uncertain; she seems to totter towards the left. The knee-jerks and other reflexes are normal. The pupils are normal. There is distinct commencing optic neuritis in each eye.

Progress of Case, November 1.—Although occasionally noisy and excitable, the patient has on the whole improved, the attacks of restlessness soon yielding to bromide and chloral. The pupils have been somewhat dilated.

November 17.—Up till yesterday there was marked improvement, mental and physical, and sedatives have hardly been needed. [Since October 28 the patient has been taking pot. iodid. and liq. hydrarg. perchlor.] She has been free from headache, and has become fairly rational. Pulse varies, 70-100. Last night the temperature rose to 101.4°, but is 97° this morning. To-day she had a fit. When seen in it she appeared quite unconscious; the left side of the mouth was drawn up, the eyes were turned to the left, the left hand was clenched, but the arm rigidly extended, as was the left leg, which twitched occasionally. The tongue was not bitten, neither were the sphincters relaxed. The attack lasted about three minutes, and was followed by bi-temporal headache.

November 23.—Another similar fit occurred early this morning, When visited four hours later she said she felt "queer," "headachy," and as if about to have another fit. She then appeared gradually to lose consciousness, and could not be roused for four minutes. There were no tonic spasms, though the left arm moved in a purposeless manner. The pupils were medium-sized and equal. Pulse = 82; respirations = 17. On the return of consciousness the patient vomited food, the pulse rose to 132, and the pupils became temporarily dilated. She was found to have double ankle clonus, and loss of tactile sensibility on both legs, but not on the arms or face.

November 28.—There have been three more fits, followed by sickness. Between the attacks the patient's mental condition is calm and rational, in fact nearly normal. The pupils are normal, the knee-jerks of normal vigour, the ankle clonus is no longer obtained and the anæsthesia of the legs has disappeared. There is a doubtful slight paresis of the left angle of the mouth. No deviation of the tongue. Right grip = 40; left grip = 35. She walks well and steadily.

There is well-marked double optic neuritis, most intense in the right eye, but the vision is good, as are the other special senses. The temperature has been normal.

December 8.—The mental and physical improvement has continued. The patient is quiet and intelligent, and though still obviously nervous, not more so than she has always been. She has gained 7lbs. in weight, is free from headache and sickness, and has had no more fits. The exophthalmos is less, though still present: P. varies from 90-120.

The optic neuritis is distinct, but the effusion is slightly paler.

December 9.—To-day the patient was sent home.

February 12, 1894.—During the two past months the patient has been seen occasionally, and on the whole the improvement seemed to be maintained, though domestic affairs at times brought out a latent irritability. Except, however, that the optic neuritis persisted there was neither sign nor symptom of serious organic disease of the brain.

It was, therefore, a surprise to hear that this morning she had suddenly died. I immediately went to her residence and though I could get no satisfactory information as to the exact mode of death, obtained permission to open the head. When this was done there was found between the cerebral hemispheres a tumour of the size of a Tangerine orange, springing from the middle of the corpus callosum and bulging slightly into each lateral ventricle. The growth was of a greyish colour and very soft. On microscopical examination it proved to be an oval-celled sarcoma. Its margins were well-defined, and the rest of the brain was healthy.

Commentary.

It will probably be conceded that an exact diagnosis of the position of this tumour during life was hardly possible. Indeed, the diagnosis of cerebral tumour at all, although inclined to by me, was very uncertain. The history of family and personal neurosis, of a somewhat dissipated life culminating in attacks of epilepsy, great mental excitement and temporary mania, suggested a generalised degenerative process in the cortex likely to lead to dementia or possibly general paralysis of the insane. The exophthalmos and rapid pulse further suggested a development of Graves' disease, which is so frequently met with among the inmates of asylums. The absence of vomiting (except after fits), the comparative slightness of headache, and the very marked mental and physical improvement which took place in hospital, all seemed to negative the view of the existence of such a coarse brain lesion as tumour. It was only the persistence of well-marked optic neuritis which caused one to look upon the improvement as but temporary and to bear in mind the probability of the recurrence of serious symptoms. Yet this fact alone could not justify an entirely unfavourable prognosis, for cases not a few have been recorded in which optic neuritis has been found without grave brain or kidney mischief, and which have made good recoveries. I have myself observed carefully the case of a young man who suffered from very severe vomiting, headache, and intense optic neuritis, who, in the course of his illness, had vertigo and epileptiform attacks, and in whom renal disease, syphilis, and plumbism could be almost certainly excluded, who yet made a complete recovery, and is now, two years later, apparently in perfect health. But such cases are rare. In young women "benign" optic neuritis is usually associated with severe anæmia, and the present patient showed no signs of anæmia or syphilis. I therefore felt it probable, when she went home apparently on the high road to health, that disease was still there and that before long we should see her again.

With regard to the relation of symptoms to the position of the growth, many points of interest arise.

I originally took the patient into hospital in order to try to localise the lesion causing the fits with a view to possible operation, and the details of the convulsions were therefore accurately noted. In the few fits seen by us in hospital the spasms were definitely one-sided, and in others, observed by her friends before admission, a distinct march of events was noticed—arm, leg, face—facts which suggested a sub-cortical lesion near the "motor" area and probably nearest to the arm centre. But the attacks after admission were very few, and there was an almost complete absence of disturbance of motor or sensory power in the affected limbs between the attacks. [Only for a day or two did there appear a very slight and doubtful weakening of the muscles on the left side of the face.] Such a diagnosis was therefore very doubtful, and the subsidence of the fits quite put aside the question of surgical intervention. The case becomes of considerable importance as a warning against hasty operation, showing, as it clearly does, that localised one-sided convulsions appearing in a definite order may be produced by a lesion remote from the "motor" area of the cortex or the fibres subjacent thereto. For the tumour, although it bulged somewhat into the lateral ventricles, did not infiltrate or perceptibly flatten either cortex or corona radiata. From its position, if it did irritate or destroy adjacent portions of the hemispheres, the parts most affected would have been the gyri fornicati or that part of the leg centre which comes over on to the mesial aspect, of which there was no clinical evidence.

The probable conclusion is that the epileptic spasms so closely resembling those usually caused by irritation of the cortical grey matter about the fissure of Rolando were in this case due to excitation of the fibres of the corpus callosum.

This view is strongly supported by the interesting experiments of Mott and Schafer,[1] who found that electrical excitation of the longitudinally bisected corpus callosum produced localised movements on the side opposite to the hemisphere with which the stimulated fibres are connected, the muscular contractions occurring as the electrodes were moved from before backwards successively in eye, head, arm, trunk and leg. They further found that if the grey matter of the connected Rolandic cortex were destroyed, these movements could no longer be obtained, although stimulation of the exposed corona radiata was still effective. From which facts they drew the probable inference that the callosal fibres were connected with the cells of the Rolandic area.

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[1] BRAIN, part ii., 1890.

To the subject of the function of the fibres I shall return later. At present the clinical fact may be noted that their excitation may produce phenomena hardly, if at all distinguishable from those consequent on excitation of the "motor" area of the cortex—a further proof of the need of caution in advising operation in cerebral disease.

The symptoms in this case differed much from those described by Dr. Bristowe,[1] whose summary has been copied into text-books as diagnostic of tumours of the corpus callosum.

A comparison, however, of his cases with this and the few others of which I have found records, hardly seem to me to support his conclusions. Dr. Bristowe's two first characteristics—(1) The ingravescent character of the symptoms; (2) The gradual coming on of hemiplegia resembling that due to hæmorrhage or embolism—certainly do not distinguish a callosal growth from one in the white substance of the hemisphere or in the corpus striatum, and the same may be said of his two last points,—"the absence of implication of the oculo-motor nerves and of direct implication of other cerebral nerves, and lastly, death from coma." His fourth symptom—"the supervention of stupidity associated for the most part with extreme drowsiness, a puzzled, enquiring look when awake, a difficulty of getting food down the throat, and cessation of speech"—is certainly met with in other cases of brain tumour and was absent in my and in other cases of growth in the corpus callosum. Dr. Bristowe's third characteristic—"the association with the paralysis of one side, of vague hemiplegic symptoms of the other,"—when present is doubtless of value, but it may, as in my case, be totally absent. It is possible, however, that the double ankle clonus and partial anæsthesia of both legs, which occurred for a short time after one of the fits, was a point of some importance as indicating a lesion to some extent affecting both hemispheres.

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[1] Brain, October, 1884

Bruns[1] concludes that:—(1) When the symptoms of an organic brain lesion are present and these symptoms increase slowly and steadily as do those of a tumour; (2) when hemiparetic or paraparetic affections are superadded; (3) when well-marked dementia, to some extent contrasting with the slightness of the general tumour symptoms (headache, vomiting, convulsions, optic neuritis) is present; and (4) when no other symptoms exist which allow of another localisation, then a tumour of the corpus callosum is probable.

But it is obvious, and Bruns himself admits it, that tumours in other situations may produce the same combination of symptoms, and also that the symptoms of a growth in the corpus callosum may vary considerably according to the part involved.

I have described (*Lancet*, July 1, 1893) a case of tumour of the caudate nucleus in which the symptoms closely resembled the above combination.

Appended (p. 548) is a tabular analysis of eleven cases of tumour of the corpus callosum, from which some general conclusions may be drawn. From this table it will be seen that Bruns' diagnostic group of signs is but rarely well developed. Thus the evidence of tumour may be slight or absent.

Headache was absent in five of the eleven cases, and was well marked only in two.

Vomiting occurred only in one (excluding my case, in which it only occurred occasionally after a fit).

Optic neuritis was noted only in three and was stated to be absent in four.

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[1] *Berl. Klin. Woch.*, 21, 1886.

The only sign of cerebral disease common to all (excepting one case of Glaeser,[1] of which the account is brief and imperfect, was *dementia*.

Dementia, in the form of monomania, was the only sign of brain disease in Berkley's[2] case, and in Oliver's[3] case visual hallucinations associated only with loss of colour vision and amblyopia suggested the diagnosis of hysteria; and in one of Brun's cases the dementia and paralytic symptoms were such as to lead Hitzig to diagnose general paralysis of the insane. In most cases it was the first symptom noted. Usually there was general failure of the intellectual powers, but in Berkley's case, except for his monomania, the patient was fairly acute. In all the slowly developed cases excitability was marked, maniacal outbreaks being common; but in my case the mental condition improved remarkably during the last two months of life.

Convulsions were present in two of the eleven cases (Glaeser, Ransom). In Glaeser's case there was left hemiplegia, with spasms of the paralysed limbs, but general epileptiform convulsions were the earliest symptoms of disease. In my case epileptic fits occurred as early as two years before death, in association with general excitability and occasional headache. The convulsions usually became general, but were more marked on the left side. It is of interest to note that in my case the head turned to the left, while in Glaeser's it turned to the right. [See Gotch and Parker, *British Medical Journal*, May 27, 1893, for the difference between men and monkeys in this respect.] My case further differed in that no paresis occurred in the convulsed parts, and the *post-mortem* examination showed no invasion of the motor path, which in Glaeser's case was apparently affected by the extensive growth.

Paralysis.—Some degree of paralysis was observed in seven of the eleven cases, and in six of these it took the form of hemiplegia, usually with some rigidity. In the hemiplegic cases the leg was earlier and more markedly affected than the arm, and in one the face escaped. Aphasia was especially noted in one, and in another the speech was thick, like that of a general paralytic. In one of Dr. Bristowe's cases, left hemi-paresis was associated with some weakness of the opposite side.

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[1] *Berl. Klin. Woch.*, 1883, p. 809.

[2] *Amer. Journ. of Med. Sci.*, 1890, p. 578.

[3] Ref. in *Lancet*, August 22, 1891.

In one case (Glaeser) there was paraplegia, the weakness and rigidity being more marked in one leg than the other. In this case, the notes of which are unfortunately meagre, the loss of power in the legs appears to have been the only symptom.

In all the cases with paralytic symptoms it was found that the tumour had invaded the white substance of the hemispheres, especially that opposite to the paralysed side. When there was no loss of motor power, either the tumour was small and did not infiltrate the centrum ovale, or it was situated at the anterior or hind end of the corpus callosum.

Sensation was less usually affected than motor power. In five of these cases it was found normal, and in two only was hemi-paresis associated with slight hemi-anæsthesia or hemi-analgesia. In the case with slight right hemi-anæsthesia (Bristowe) the growth in the anterior half of the corpus callosum extended some distance into the left centrum ovale; in that with slight right hemi-analgesia, the tumour was situated just in front of the splenium and extended slightly into the right precuneate lobule and rather more into the left central convolutions. The gyri fornicati were free, but the left optic thalamus was somewhat atrophied.

In one case only (Oliver) was the special senses markedly affected. Here vision became gradually lost in the left eye, while colour vision was impaired in the right; hearing was diminished in the left ear, and taste and smell were impaired. Visual hallucinations were the prominent feature.

The tumour in this case is described as having arisen from the knee of the corpus callosum, and pressed downwards on the optic nerves as well as on the uncinate gyri.

It would thus appear that the interference with vision was due to direct damage to the nerves, and not to injury of optic radiations or cortical visual centre.

The *reflexes* do not call for special comment, except that there seems rather less tendency to increased tendon reflexes in the paretic limbs than might be expected with spastic hemiplegia. In only two cases were the knee-jerks found increased on the paralysed side, and the only occurrence of ankle clonus seems to have been what was found in my case just after a fit.

The pupil reflexes seem to have been normal, except in Oliver's case, of pressure on the optic nerves.

The *cranial nerves* almost invariably escaped; the only change noted being inequality of the pupils in three cases—including that of Oliver. But this inequality, as is well known, may be produced by pressure on the cortex, without involvement of the nerves at the base of the brain.

The *duration* of symptoms of disease varied from five weeks to three years.

Death occurred in five cases from gradual coma, usually associated with pyrexia; in two cases from multiple hæmorrhages, in two from pulmonary complications, and in two the end was sudden but of unexplained mechanism.

Diagnostic Conclusions.

From this analysis it appears doubtful whether a positive diagnosis of tumour of the corpus callosum can ever be made. The symptoms may closely resemble those not only of tumours of the central ganglia, centrum ovale, or frontal lobes, but also those of softening from thrombosis of the Sylvian artery and those of general paralysis of the insane and of other forms of insanity.

It appears that a tumour in this position can exist for long and attain considerable size without producing marked pressure symptoms. The tumour symptoms are generally slight. Paralysis may follow the invasion of a hemisphere by the growth, and usually takes the form of hemiplegia with rigidity, and is associated with little or no anæsthesia. The leg tends to be affected more than the arm, and the arm than the face. Occasionally the opposite limbs may share in the weakness, and then the diagnosis is greatly facilitated, as only cerebral softening, multiple tumours, or growths in the pons or medulla would be likely to produce the same combination.

Early and marked mental degeneration would appear to be a characteristic symptom. The cranial nerves are not affected. Convulsions may or may not be present, but are of little localising value. Vertigo and ataxy are absent.

To sum up, a tumour of the corpus callosum may be diagnosed as probable when there are present:—

(1) Gradual but marked mental change, in more acute cases taking the form of increasing stupor, in the more chronic appearing as insanity with delusions, irritability, and maniacal outbursts.

(2) Moderate development of tumour symptoms (headache, optic neuritis, vomiting).

(3) Hemiparesis with rigidity, with or without slight hemi-anæsthesia, and with or without convulsions.

If the opposite limbs are also slightly affected, the diagnosis is strengthened.

(4) Or convulsions without paresis—the spasms being bilateral, but more intense on one side.

(5) Integrity of the cranial nerves.

(6) Little or no change in the tendon reflexes.

This group of symptoms may, however, obviously be produced by other cerebral affections, and it is only when the paresis or spasms affect to some extent both sides, without other evidence of a multiple lesion, that any degree of certainty can be attained.

Relation of Symptoms to Function of Corpus Callosum.

Our knowledge of the anatomical and functional relations of the corpus callosum is at present so slight that but few conclusions can be drawn as to the dependence of the above described symptoms upon the lesion of that structure.

Koranyi[1] has shown that section of the corpus callosum of dogs produces no permanent paralysis or notable disturbance, though convulsions of the whole body may at times occur. I am not aware that these experiments have been repeated on monkeys. Several instances of its complete absence in man have been recorded, in which the symptoms during life presented considerable variations. Thus Sir James Paget[1] found this body absent in a patient "not remarkable for any excellence or great defect of mind." Langdon Down,[2] in the brain of an idiot who could walk and run and say a few words, found absence of the corpus callosum, body of the fornix, and the soft commissure. Dunn[3] found no corpus callosum but an otherwise well-developed brain in the body of a man of whom there was no probability that he had been an idiot. In this case the body of the fornix was also absent.

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[1] *Pflüger's Archiv*, Bd. xlviii.

Dr. Alexander Bruce[4] has given a full account of a case of his own, in which the corpus callosum was absent without marked mental defect, and has also made a careful digest of fifteen other cases of complete absence of this body, as well as others in which it was primarily defective and others in which its absence was apparently secondary to other cerebral lesions. The study of these cases leads to the conclusion that absence of the corpus callosum alone need not cause mental or bodily disorder, but that such are due to other associated brain defects. Dr. Bruce points out that the recorded facts lend no support to the anatomical and physiological doctrine of Meynert, that this structure co-ordinates corresponding convolutions in the two hemispheres, and that, in fact, this view is purely theoretical. So far as I know, the only experiments giving positive results on the function of the corpus callosum are those above referred to of Mott and Schäfer.[5] These clearly show that impulses may travel along the fibres of this body up to cells of the Rolandic convolutions, and excite these cells to liberate motor currents downwards. But they do not show with what structures these fibres are connected at the end distal from the Rolandic cells. Dr. Mott[6] has, however, in his report on this work to the British Medical Association, given a diagram in which this distal end terminates in a ramification over a corresponding

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[1] *Med. Chi. Trans.*, vol. xxix.

[2] *Med. Chi. Trans.*, vol. xliv.

[3] *Guy's Hospital Reports*, vol. xlvi., 1889.

[4] BRAIN, July, 1889.

[5] *Ibid.*, 1890, p. 174.

[6] *Brit. Med. Journal*, May 17, 1890.

"motor" cell in the opposite hemisphere—an arrangement which apparently depends merely on Meynert's theory.

If we seek information from the study of secondary degeneration, we find from the experiments of Sherrington[1] that lesions of the cortex, not only of the cord area, but of areas anterior, posterior and lateral to it, are followed by a degeneration of scattered fibres in the opposite hemisphere, their distribution not being further indicated. In Boyce's[2] experiments, after the removal of the whole of one cerebral hemisphere of a cat, degenerate fibres were traced across the corpus callosum into the other hemisphere which tend to turn *upwards*. Neither of these series of experiments teaches us the nutritive centre of the bulk of the callosal fibres, the normal direction of impulses along them, or their actual anatomical relations to the cortical cells. According to Mott's diagram, a callosal fibre is a process of a cortical cell in one hemisphere, ending in a dendron over a cortical cell in the other hemisphere; but the actual facts at our disposal give us no exact information as to one end of the fibre or the other. The comparatively small number and scattered distribution of fibres which degenerate after even extensive experimental lesions of the cortex, is in agreement with what is found in human brains after softening of the Rolandic cortex, where little or no degeneration has been traced across the corpus callosum, but is hardly compatible with the view that most of these fibres are processes of cortical cells.[3]

The alternative view, therefore, presents itself that some, at least, of the callosal fibres have their nutritive centre below the cortex. The case of tumour of the corpus callosum recorded by Berkley, appears to lend support to this view. Here a small growth destroyed most of the fibres just behind the genu; the degeneration could be traced into the frontal lobes, and there was marked wasting of the frontal convolutions. One may conceive that, as in amyotrophic lateral sclerosis, the cells of the anterior cornu degenerate after destruction of the terminal ramifications of the pyramidal fibres, so the cells of the frontal cortex shrink after atrophy of fibres of the corpus callosum.

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[1] *Journ of Physiol.*, vol. x.

[2] *Brit. Med Journ.*, Sept. 23, 1893.

[3] I have cut sections of the whole of the corpus callosum from a case of hemiplegia of two years' duration, in which there was marked softening of the left Rolandic area due to arterial thrombosis. Though there was well-marked descending degeneration in the left internal capsule and the crossed pyramidal tract of the cord, readily visible to the naked eye, in the corpus callosum one could only distinguish microscopically, in sections stained by Weigert's method, a few scattered degenerate fibres.

These considerations lead us to the views of Hamilton,[1] which have been expounded in a number of papers for several years, but which appear to have met with but little acceptance. Using the anatomical method, Hamilton arrived at the conclusion that the callosal fibres are not commissural for corresponding parts of the hemispheres, but that, starting from the cortex on one side they cross to the other hemisphere, arch down into the anterior limb of the inner capsule, and to some extent into the outer capsule, and end, for the most part, in the optic thalamus, in the cells of which they may possibly have their nutritive centre. The tracing of the path of fibres in the centrum ovale by the anatomical method alone is undoubtedly a task of great difficulty. It is quite possible, therefore, that, as Bruce suggests, some of Hamilton's arched "callosal" fibres may really descend from the gyrus fornicatus and not from the corpus callosum, but the evidence adduced by Hamilton appears too strong to be completely set aside. If callosal fibres have their trophic cells in the thalamus, then destruction of the corpus callosum need not lead to atrophy of the internal capsule, as has been argued against Hamilton by Bruce. The degenerations found in Berkley's case might, however, naturally follow. The most serious argument against the doctrine of Hamilton appears to be the statement (made by Bruce) that in the human infant up to three months the callosal system is non-medullated, while "almost the whole of both limbs of the internal capsule are medullated." Hamilton, however, speaks of the arching downwards of the corpus callosum as being very well seen in the four months human embryo. He also found the callosal arch naturally dissected out in a porencephalic brain. Finally, in the brains of certain lower mammalia, such as the armadillo, the corpus callosum has been figured by Rabl-Rückhard as turning downwards into the inner and outer capsules.

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[1] *Proc. Roy. Soc.*, 1884; *Journ. of Anat. and Physiol.*, 1885; "Text-book of Pathology, 1894. [See also BRAIN, vol. viii. (1886), p. 145, and Beevor's criticism, *ibid.*, p. 377.]

Hamilton's anatomical views, therefore, cannot be said to be overthrown, but on the contrary, have some amount of probability.

The stimulation experiments of Schäfer and Mott are in harmony with them. We may thus consider some of the fibres of the corpus callosum as a part of the afferent system, as conducting impulses upward, and those fibres which go to the Rolandic area may be looked upon as the afferent part of the kinæsthetic mechanism of cells and fibres. Those fibres which are connected with the frontal and occipital lobes, on the other hand, may conduct some of the stream of afferent impulses which excite perceptive and ideational cerebral processes. There are many grounds for considering the optic thalamus as one of the halting-places of sensory impulses. The specialised senses, such as touch and sight, are known not to travel by the anterior limb of the internal capsule on their way to the cortex, but their impulses form but a comparatively small proportion of the multitude that stream upward. The path by which currents from the cerebellum travel from the basal ganglia to the cortex, the paths of afferent impulses from the viscera and the muscles, and of the vague tactile sense which is often lost when the "motor" cortex is destroyed, may lie among these callosal fibres. The results of degeneration experiments make it further probable that these fibres do not actually join the cells of the cortex, but probably end in a ramification among the processes of those cells, as it is supposed do the pyramidal fibres of the cord with the protoplasmic processes of the cells of the anterior cornua.

Such a view of the structure and function of the corpus callosum is in harmony with the facts observed in cases of disease of that organ. A disordered sensorium is a prime condition of mental aberration; upon the regulated stream of sub-conscious impressions depends the order of conscious processes. A tumour in the corpus callosum on this theory will now diminish, now increase to violence the force of this stream, and dementia or mania ensues. If the lesion irritate also those fibres ascending to the cells of the Rolandic or "cord" area of the cortex, violent action of those cells is produced and convulsions ensue. What happens when the growth destroys these fibres without involving the hemispheres as well is rather uncertain. Possibly some of the paresis observed may be thus caused; but Koranyi's experiments seem to show that in dogs, at least, the functional activity of the cells does not entirely depend on the integrity of these fibres, and in the reported cases of brain disease there has usually been too much involvement of the white substance of the hemispheres to allow of making deductions on this point. Experiments on monkeys are desirable to throw light on this point. One would expect to find after section of the corpus callosum in the monkey some paresis and affection of the muscular sense, which would probably be, however, only temporary. Experiments are also needed to study the degenerations consequent on destruction of the optic thalamus.

Pathological observations have so far thrown but little light on the relations of the optic thalamus to the cortex or to the corpus callosum. Though hyperæmia and small hæmorrhages have been often noticed in the thalami in cases of chorea, yet it is not certain that they are more abundant there than in other parts of the central nervous system. Hammond, in his original description of athetosis, attributed this disorder to a sclerosis of the corpus striatum and optic thalamus, and Charcot[1] found the posterior parts of the optic thalamus and corpus striatum affected in three cases of post-hemiplegic choreoid spasms; but the latter goes on to say that he has "often and often seen the optic thalamus and lenticular nucleus affected in their several parts with the most varied lesions and not the slightest trace of choreic movements resulting." In one case of

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[1] *Lectures* (Syd. Soc., second series.)

"pre-hemiplegic chorea" Charcot found a small hæmorrhage in the posterior half of the optic thalamus.

Dr. Gowers[1] has recorded a case of right hemiplegia, recovered from in three months but followed by ataxy of the right arm, in which the only brain lesion found was a cicatrix in the left optic thalamus.

In this case, as in the last quoted one of Charcot, it appears probable that the lesion in the thalamus disturbed the adjacent motor fibres in the internal capsule. If the symptoms were produced by disturbance of *afferent* impulses, these cases indicate that early thalamus is chiefly connected with the cortex of its own side of the brain, and not, as it should be on Hamilton's theory, with the opposite hemisphere. At any rate the very interesting case of Dr. Gowers lends no support to Prof. Hamilton's view.

It is much to be desired that in any future case of a similar nature, a careful histological study of fibres both above and below the thalamus should be made.

It is certainly remarkable that so little degeneration should cross the corpus callosum in cases of even old and extensive destruction of the "motor" cortex; but it is by no means as yet proved that the nutritive centre of its fibres is below. Probably it will be found that the fibres forming this structure are of mixed origin and function.

POSTSCRIPT.

Since the above paper was written, another case has been reported by Francis and Starr in the American *Journal of the Medical Sciences*, June, 1895.

The patient was a woman aged 45. The duration of the case was fifteen months. During the first nine months the only symptoms were four attacks of general epileptiform convulsions. At the tenth month there was vomiting for a week, and after this progressive mental and physical exhaustion set in. By the twelfth month there was great mental dulness, she was too weak to sit up, was totally blind and had double optic neuritis. The head was persistently deviated to the right, the right pupil was dilated

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[1] *Med. Chi. Trans.*, vol. lix.

Name of Observer.	Age and Sex.	Duration of Symptoms.	Tumour Symptoms.			Mental State.	Convulsions.
			Headache.	Vomiting.	Optic Neuritis.		
Bristowe	♂ 46	14 weeks	The first symptoms	Absent	Not stated	Gradual failure, drowsiness. No excitement	Absent
Bristowe	♂ 41	5 weeks	Moderate	Absent	Present	Slow growing stupor	Absent
Bristowe	♂ 51	12 weeks	Absent	Absent	Absent	Increasing drowsiness	Absent
Glaeser	♂ 72	9 weeks	Absent	Absent ?	?	?	Absent
Glaeser	♀ 30	12 weeks	Slight	Absent	?	Some loss of memory	The first sympton repeated, at firs general then left sided, but hea turned to right
Glaeser	♀ 60	4 months	Severe at times	Frequent	Present	Irritability, gradual failure, delirious mania	Absent
Berkley	♂ 45	Some years	Absent	Absent	Absent	Insanity, monomania the only symptom	Absent
Oliver	♀ 43	3 years	Absent	Absent	Absent	Visual hallucinations	Absent
Ransom	♀ 24	2 years	Severe at first, afterwards absent	Occasionally after a fit	Present	Excitability, mania. Great improvement for 2 months before death	Early and frequen mostly left-sided head and eyes turned to left
Bruns	♂ 77	2 years	Present	Absent	Absent	Progressive dementia with occasional mania	Absent
Bruns	♂ 49	3 months	Absent	Absent	?	Dementia, irritability, symptoms suggesting general paralysis of the insane	Absent

Note.—The fourth case of Dr. Bristowe and the third of Bruns are not included in this list,

Motor Power.	Sensation.	Reflexes.	Cranial Nerves.	Mode of Death.	*Post-mortem* Examination
Right hemiparesis	Slight failure of vision?	Right knee-jerk increased	Normal	Coma. Fever on day of death	Tumour involving nearly whole of corpus callosum and reaching to left internal capsule
Right hemiplegia, with rigidity, aphasia	Slight right hemianæsthesia	Right knee-jerk increased	Unequal pupils, varying	Coma, with fever for four days	Growth in anterior half of corpus callosum spreading into corresponding part of hemispheres, more into left, internal capsules free
Left hemi-paresis, but right limbs also weak	Normal	Normal	Normal	Coma, with slight fever just before death	Sarcoma on anterior ¾ of corpus callosum and fornix and invading each centrum ovale above corpus striatum, slightly more extensive on right side
Rigidity and weakness of legs, more of left	Normal	Normal	Normal	Bed-sore and broncho-pneumonia	Tumour = small apple, in genu of corpus callosum invading white substance of hemispheres
Left hemiplegia, but frequent spasms of paralysed limbs	Normal	?	Normal	Very sudden	Large tumour of corpus callosum growing far into right hemisphere
Sudden paresis of right leg, followed by face and arm of same side	?	?	Normal	Coma	Tumour of hinder end of corpus callosum growing very slightly into right and rather more into left hemisphere
Normal	Normal	Normal	Normal	Œdema of lung	Tumour = filbert just behind genu of corpus callosum. Wasting of frontal lobes
Normal	Amblyopia in left eye; colour sense impaired in right, smell lost; taste impaired. Hearing impaired in left ear	Normal except left pupil immobile to light	Normal except for left pupil	Gradual coma	Tumour of genu corpus callosum passing on uncinate gyri, and downwards on optic nerves
Normal	Normal	Normal except after a fit	Normal	Sudden	Tumour = Tangerine orange growing from middle of corpus callosum and pressing on hemispheres slightly
Right hemiparesis with rigidity; face not involved	Partial analgesia on right arm and leg	Normal	Normal	Cerebral hæmorrhage	Gliosarcoma, 3 cm. long, just in front of splenium, bulging slightly into right precuneate lobule, and rather more into left central convolutions; gyrus fornicatus free. Left optic thalamus somewhat atrophied. Atheromatous arteries, hæmorrhage in lateral ventricles; senile atrophy of cortex
No paralysis or spasm. Speech thick	Absent	Normal	Unequal pupils	Cerebral hæmorrhage	Tumour of splenium infiltrating both precuneate lobules and growing into posterior cornua of lateral ventricles. Also growths in right temporal lobe, right Island of Reil, and right optic thalamus Multiple capillary hæmorrhages

as in each the lesion of the corpus callosum was but a part of more extensive disease.

and there was right internal strabismus. There was no other paralysis and no affection of general sensation. The knee-jerks were normal. Headache was throughout slight. Increasing stupor led to death.

At the autopsy was found a large glio-sarcoma rising from the anterior knee of the corpus callosum and infiltrating the greater part of each frontal lobe. It did not appear to compress the optic chiasma.

It will be noted that this case differs from most of the others in the affection of ocular nerves.

Although it apparently grew from the corpus callosum, its subsequent extension makes it more correctly spoken of as a tumour of the frontal lobes.

MULTIPLE TUMOURS OF THE BRAIN; FIBRO-CYSTOMA OF PONS AND CEREBELLUM AND MULTIPLE FIBRO-PSAMMOMATA OF DURA, PIA-ARACHNOID AND CORTEX CEREBRI.

BY F. W. LANGDON, M.D.

Neurologist to the Cincinnati Hospital, and to the Ophthalmic Hospital of Cincinnati.

I SAW Miss X., aged 32, brunette, by request of Dr. W. O. Smith on January 15, 1895. Her father and mother were living at advanced age and in good health. Two brothers and two sisters, all of adult age, were living and in excellent health. Two sisters died in infancy; cause unknown. No history of nervous disease or of tumours anywhere in the family.

Personal History.—Patient is one of twins, both girls. She has had measles and perhaps other children's diseases, otherwise enjoying good health until seven years ago, when she experienced a severe attack described as "cholera morbus." Was sick several days at this time and since then has had more or less ill-health, described as "nervous trouble," for which she has consulted various physicians. In November, 1890, she consulted Dr. S. C. Ayres, to whom we are indebted for the following notes: "I first saw Miss ——, in November, 1890. She was deaf in both ears. It seemed to be a case of chronic aural catarrh. There was no obstruction of the Eustachian tubes and but little alteration in the appearance of the drum membrane. Treatment did not relieve her and was not continued long. She had at this time sick headaches. I examined her refraction and found that she had compound hypermetropic astigmatism in both eyes, and gave her correcting lenses; her vision was perfect. On February 27, 1892, I saw her in consultation with Dr. Zenner on account of a marked papillitis of both eyes. Her vision was 0·9, right, and 0·7, left. Two days later I saw her again, and then there was a remarkable and unusual phenomenon in the right eye. In looking at the test card, vision would drop from 0·7 to 0·4 and then in a moment or two rise to 0·7 again. I watched this change for some time while she remained in my office. No such change was present in the left eye. A perimetric examination showed that the upper part of the field of vision was contracted and also the field for red. I did not see her again until January 28, 1895; she was then suffering so much pain that I could not test her accurately, but I found that her central vision was normal as she could read fine print with either eye. I found, however, that there was paresis of the right abducens, with diplopia on the right side."

Patient states (January 15, 1895) that she had within a few months past, several attacks of vertigo and vomiting, apparently brought on by sudden movements, as turning in bed, &c. Says she *feels nausea before vomiting*. Also complains of numbness of left extremities and left side of body.

Clinical History.—Patient in bed, but has been up and about room more or less daily. Decubitus dorsal. Temperature under tongue 99·4° F.

Nervous System.—Mental state normal; memory excellent; patient converses and writes with intelligence and manifests much interest in her own case. Speech somewhat slow but unhesitating and correct. Handwriting steady, uniform and legible; letters well formed, words correctly spelled and sentences properly constructed.

Sensory Symptoms.—She complains of a general sense of fatigue and excruciating paroxysmal pains in the head, chiefly in right parietal and occipital regions, from which she derives relief by assuming an opisthotonic posture (resting head on vertex). Examination also reveals hyperæsthesia of the scalp over the right frontal, parietal and occipital regions. Tactile paræsthesia of slight degree over left face, body and extremities.

Vision: Pupils moderately dilated, responsive to light. Patient complains of double vision at times, but reads and writes without apparent difficulty. (See previous report.)

Hearing markedly impaired on both sides, practically absent on left. (See also previous report.)

Taste and smell not tested, but no deficiency or perversion noted by patient.

Motor Symptoms.—Left hemiparesis of face and limbs present, and patient says she tends to turn toward left side in walking. Tongue protruded in median line. Later observations (within a month) by Drs. P. S. Conner and W. O. Smith, record a marked deviation to left, which disappeared within a few days to return later. During the last few days of life, marked symptoms of bulbar paralysis developed—as evidenced by great difficulty in swallowing and articulation.

Patellar and olecranon reflexes not notably altered.

Heart sounds weakened, no bruit detected. Pulse 84 to 90, soft, quick stroke.

The other systems were normal, and urinary analysis showed no abnormal constituent.

The salient features of the case at the time of my first and only visit (thirty-nine days before death) were:—Violent paroxysmal pains in head, hyperæsthesia over right side of scalp, diplopia, bi-lateral deafness, left hemiparesis and vomiting *with nausea*, added to which were the optic neuritis and abducens paralysis, noted by Drs. Zenner and Ayres. Upon these I based my opinion of a tumour at the base of the brain, probably pressing on the pons varolii and involving the fourth, fifth, seventh and eighth nerves or their nuclei. From these symptoms, especially the left hemiparesis, I supposed that the growth was in the upper half of the pons on the *right* side, whereas its actual situation discovered after death was over the lower half and *left* side.

It was plain that no one cortical lesion would account for all the symptoms presented; and the absence of convulsions, monoplegia or mental impairment excluded, to my view, an extensive or multiple cortical involvement. Further, the evidence pointed to disturbance of the right fourth and sixth nerves (or their inter-communications) to the right fifth, left seventh and both eighth nerves also; and only in the region of the pons was this possible to occur from a single lesion, without loss of consciousness.

The vomiting and rotatory gait would naturally point to the cerebellum, but might easily be accounted for by irritation of its peduncles by a growth in the pons.

Report of Autopsy, February 28, 1895, at four o'clock, p.m., five days after death.

Body well preserved; no embalming fluid or other preservation used. Rigor mortis present to a slight degree. The head only was examined. Calvarium removed in the usual manner. Dural outlines and general consistence of brain normal. Dural vessels markedly hyperæmic, the larger meningeal trunks standing out as prominent dark blue cords. Cortical veins appear through dura fuller and more distinct than normal.

The lower border of falx cerebri at a point 6 cm. posterior to the crista galli contained an ovoid neoplasm, the size of a kidney bean, enclosed within the layers of the falx; the left layer, however, had undergone absorption, so that the fibrous capsule was deficient over the central two-thirds of the growth on the left side. Immediately posterior to this growth the falx in the region of the inferior longitudinal sinus, for the space of 25 mm. longitudinally by 5 mm. vertically, presented a recticular or cribiform appearance apparently the result of imperfect development or absorption; but this bore no direct relation to the growth mentioned, nor to any other.

A second neoplasm, half the size of the first, of similar appearance but more flattened, was situated at the superior margin of the falx, slightly posterior to the first mentioned growth, with which it was connected by a narrow isthmus.

Three similar growths, also more flattened than the first projected from the inner surface of the dura of the convexity over the posterior extremities of the first and second frontal convolutions. These measured respectively 3 mm., 6 mm., and 20 mm., in greatest extent and from 2 to 3 mm. in thickness. A sixth neoplasm, ovoid in form and the size of a large kidney bean, projected from the under surface of the dura—but still enclosed between its layers—immediately to the right of the great longitudinal sinus, which it did not invade. This growth occupied a position nearly corresponding with the upper extremity of the Rolandic fissure.

A seventh growth, also the size of a kidney bean, was enclosed in the meshes of the pia, just anterior to the *lamina cinerea*, where it bridged the great longitudinal fissure and penetrated slightly the cortex of both frontal lobes (*gyri recti*).

The eighth and principal tumour (fig. 1) came into view on dividing the *tentorial* attachment to the petrous margin. Its anterior extremity, the size of the end of the little finger,

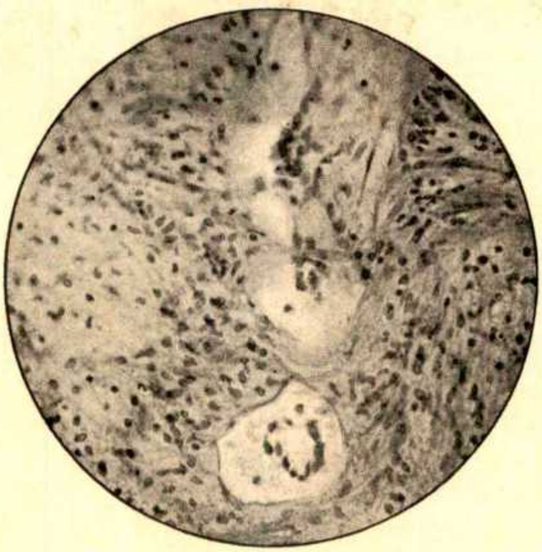

FIG. 2.

A section of solid portion of main tumour (No. 8 in report). x 320. Photo-micrograph by Dr. M. H. Fletcher.

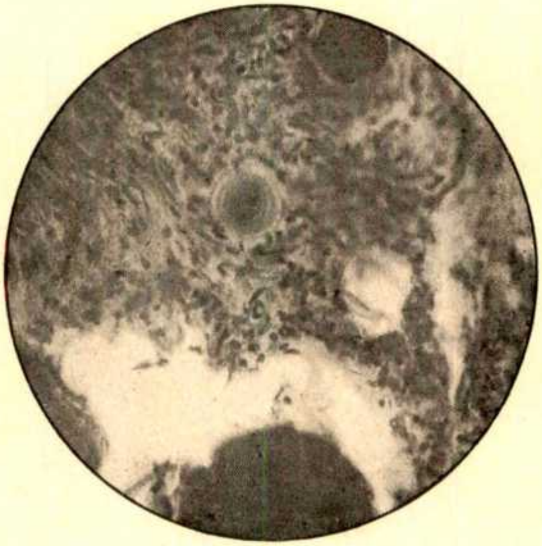

FIG. 3.

Section of tumour from falx cerebri (No. 1 in report) showing concentric laminated nodule near centre of field. x 320. Photo-micrograph by Dr. M. H. Fletcher.

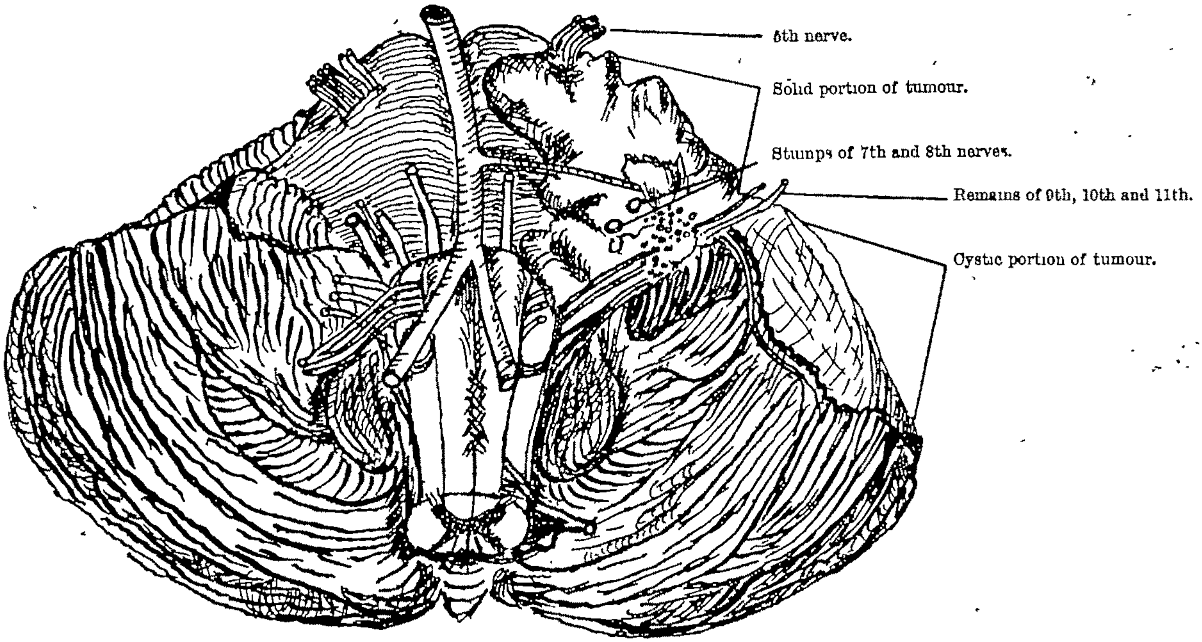
5th nerve.
Solid portion of tumour.
Stumps of 7th and 8th nerves.
Remains of 9th, 10th and 11th.
Cystic portion of tumour.

Fig. 1.
Sketch of principal tumour and surroundings—natural size, made immediately after removal.

projected from the under surface of the pons on the *left side.*

As the entire tumour came into view, it was seen to be a firm, irregularly nodular, yellowish, encapsulated mass, in bulk about equal to an English walnut, but more flattened and irregular in form.

This growth involved the left lateral half of the pontine surface—for its posterior two-thirds—and also extended outward and posteriorly, invading the antero-lateral margin of the left cerebellar hemisphere. Extending still further in the same direction, this solid portion of the growth was continuous with a cystic portion, thin-walled, evidently composed of pia and arachnoid, and containing about half an ounce of clear, amber-coloured, somewhat viscid fluid; a small portion of this fluid was decidedly gelatinous in consistency. The left cerebellar hemisphere, where compressed between the growth and the tentorium was softened and semi-diffluent.

The left half of the pons (its posterior two-thirds) was compressed and atrophied by the growth to about one-half its normal lateral extent (on its inferior surface) and *the pons as a whole was displaced to the right.*

The left flocculus was distorted, flattened and continuous with the under surface of the growth.

The considerable segment of the left cerebellar hemisphere that was atrophied and absorbed or softened by the pressure, amounted perhaps to one-fourth of the total bulk of this hemisphere.

Except as described the external appearance of the cerebellum was normal.

The cranial nerves in *apparent* relation with the growth, were the left fifth which deeply grooved its anterior (superior) surface for a distance of 11 mm., and apparently terminated in the tumour substance as a yellowish, moderately firm, mixed mass of gelatinous substance and nerve fibres. Tracing the nerve anteriorly from the growth it passed as a similar bundle of mixed gelatinous and normal nerve fibres, to the Gasserian ganglion, beyond which the fibres and divisions appeared normal. The above described conditions involved both roots of the left fifth nerve.

The left sixth nerve, somewhat displaced mesially at its origin by lateral pressure on the pons above referred to, passed forward in the groove formed at the junction of the pons and the neoplasm. It was normal in appearance and not invaded by the growth.

The left seventh and eighth nerves were not visible at their usual superficial origin since this was occupied by the growth, which is here apparently continuous with the pons and oblongata substance.

Two rounded stumps are apparent on the surface of growth corresponding to their usual course, from which the nerves have probably been torn in removing the specimen. (See fig. 1.)

The ninth, tenth and eleventh nerves emerged normally, traversed the under surface of the growth for 20 mm. and partly entered its substance, a few fibres, mainly of the tenth nerve traversing the growth, emerging laterally and continuing their course towards the jugular foramen.

The remaining fibres, by far the larger bulk of these nerves, had softened and disappeared upon reaching the growth; and in this immediate region of disappearance of nerve fibres the surface of the growth is broken into miliary nodules, perhaps a hundred in number.

The left twelfth nerve emerged normally and was not directly compromised by the growth, but was probably subject to mechanical pressure at times during life.

The left anterior inferior cerebellar artery, as shown in fig. 1, traversed the lower surface of the growth for 19 mm., penetrated its substance for 13 mm., and emerged to resume its normal distribution, apparently not infiltrated or compromised in any manner by its intimate connection with the tumour. On section the cerebral cortex and alba were of normal appearance and consistence. The lateral ventricles were markedly dilated, their anterior and posterior cornua readily admitting three fingers; choroid plexuses fringed with numerous clear round cystic dilatations, miliary in size, containing a pellucid fluid.

A brief analysis of the symptoms in the light afforded by the autopsy, may be of interest from the standpoint of localisation of function. At the outset, our attention is naturally directed to the relationship, if any, between the supposed attack of "cholera morbus" seven years previous to death and the pathological process in the brain. Was the attack of vomiting and diarrhœa to be looked upon as being in any sense an ætiological factor in the disease? Or was it symptomatic of an early stage of the growth in the cerebellar peduncles or the vagus nuclei and trunk? The latter would seem the more probable explanation to my mind.

As regards the area of nervous substance involved, the symptoms presented were less marked up to the last few days of life than might have been expected, having in view only the ordinary facts of localisation. These comparatively slight effects of so serious a lesion are to be accounted for probably by the extremely gradual progress of the growth, allowing time for a considerable degree of accommodative adjustment on the part of the tissues involved. This, however, eventually reached its limit, and hence the sudden development of marked bulbar symptoms toward the final end.

The cranial nerves and nuclei involved symptomatically correspond with the anatomical situation of the growth, as may be seen by our diagram (fig. 1), with the exception of the *fourth* and *fifth* nerves. While ocular examination by Dr. Ayres revealed an apparent paralysis of the fourth, its trunk was not directly involved in any part of its course. Hence the diplopia, which was the apparent result of its impairment, must probably be attributed to the involvement of the co-ordinating fibres (*posterior longitudinal bundle*) by which harmony of action is brought about between the nuclei of the third, fourth and sixth.

It will be noted that, though the fifth nerve was seriously compromised in structure, some symptoms that might have been expected, viz., hyperæsthesia, anæsthesia, and trophoneuroses of *left face* and its organs, with spasm, paresis or paralysis of muscles of mastication, did not occur, as sufficient fibres to carry on the function evidently escaped degeneration.

We are also confronted by the clinical symptom, in this case, of *right-sided hyperæsthesia,* over the forehead and scalp. Since the main trunk of the *left* fifth was the one apparently irritated by the growth, the hyperæsthesia would naturally be expected to be referred to its terminal distribution. The explanation of this apparent discrepancy in localisation may be due to the fact that the tumour by its pressure exerted from the left side toward the right may have pressed the right fifth nerve against the acute margin of the tentorial attachment to the petrous bone, and so given rise to the symptoms mentioned.

The bi-lateral deafness, most marked on the left, was evidently due to two factors: (1) The direct involvement of the left acoustic nerve by the growth; and (2) Pressure on the pons affecting the fibres from the right nuclei in their course toward the cortex, through the pons, crus and internal capsule.

The absence of complete left facial paralysis (there being only paresis present) is explicable by the fact that some fibres of the *portio dura*, though surrounded by the growth, maintained their power of conduction, as was also the case with the greater portion of the glosso-pharyngeal, vagus and accessorius up to the last few days of life.

The left motor paresis was apparently due to pressure on pyramidal fibres *after* their crossing.[1]

It is evident, moreover, that a very limited lesion of the *right upper* half of pons (above the facial decussation) would produce practically the same motor symptoms as here presented by our tumour in its *lower left* segment.

[[1] In the discussion which followed the reading of this paper before the American Medical Association at Baltimore, May, 1895, Dr. Charles K. Mills and Dr. H. T. Patrick very pertinently directed attention to the fact that it was difficult, even with the specimen before us, to account for the motor and sensory paresis existing on the same side as the tumour, since the latter was above both the motor and sensory decussations. It was this circumstance also which led me in the first instance (*ante-mortem*) to locate the lesion on the right side of pons. After a careful weighing of the matter in all its aspects, I have concluded that the only rational explanation, in the light of our present knowledge of the subject, is that the motor paresis and sensory paræsthesia were due to a "*contre coup*" effect, *i.e.*, of pressure by the tumour of the right periphery of the medulla against the comparatively acute margin of the foramen magnum at a point above (before) the motor decussation and also above (after) the sensory (fillet) decussations. The left *facial* (motor and sensory) symptoms, on the other hand, are sufficiently accounted for by the direct involvement of the tri-facial and the portio dura by the tumour.—(L.)]

It is worthy of note that the vomiting, which occurred chiefly or entirely upon sudden movement, was *preceded by nausea*, contrary to the rule in cerebellar vomiting. This would perhaps indicate that the *involvement* of the vagus trunk or its nuclei was the cause of the nausea and vomiting.

We may also note the protrusion of the tongue to left side (left paralysis) and its apparent recovery within a few days. If due only to direct pressure of the solid growth a right-sided tumour pressing on *upper half* of pons would also account for this symptom, by involvement of cortical tract to medulla. In this case, however, the symptom would have been permanent, whereas its sudden disappearance is accounted for by fluid pressure on the hypoglossal trunk (shifting of fluid in cystic portion of growth).

One more symptom deserves mention. The patient derived great relief from the severe intra-cranial pain by assuming a semi-opisthotonic position, resting head on vertex. It is evident that by this position the weight of superimposed brain mass was removed in a measure from the growth, and especially from the left fifth nerve, which in the ordinary position of head was compressed between the tumour and its solid backing of dura and bone below, and the brain above.

Therapeutically, Dr. Smith informs me, the greatest relief to pain was derived from *antipyrine*, its effects being superior even to morphine in this case, and without disagreeable accompaniments.

PATHOLOGICAL REPORT ON THE TUMOURS.

BY ARCH. I. CARSON, M.D.
Curator to the Cincinnati Hospital.

Microscopical Examination.

Two small pieces from different peripheral portions of the main tumour (No. 8 in report) were examined. Both specimens show practically the same conditions; that is, fibroid material arranged irregularly in intercrossing fasciculi and in some areas in whorls. One specimen was of quite close texture and not very vascular. There were present numerous small bodies which stained most deeply, round, oblong, and spindle-shaped; evidently nuclei of connective tissue cells and embryonal cells.

The second specimen was of a much looser texture and more vascular. It showed similar nuclei, but very much fewer spindle-shaped ones. The fibres were not so frequently arranged in bundles, but were interwoven in all directions. The vessels, around which the fibres were arranged more or less concentrically, were filled with red blood cells. (Fig. 2.)

From the examination of these specimens, from two different parts of the tumour, it is concluded that the growth is a fibroma.

There was also examined a small mass (tumour number one in report) ovoid in shape, measuring 10 mm. in length, 7 mm. in width, and 4 mm. in thickness, which was removed from about the centre of the falx cerebri. This mass under the microscope showed a highly cellular fibrous tissue, containing numerous concentrically arranged chalky concretions, the latter characteristic being evidenced in the cutting of the sections. It is concluded that this mass is a psammoma. (Fig. 3.)

A CRITICAL STUDY OF A FEW OF THE CHANGES FOUND IN THE FIELDS OF VISION, TAKEN WHILST THE EYES ARE PLACED AT RIGHT ANGLES TO THEIR ORDINARY POSITION.

BY CHAS. A. OLIVER, A.M., M.D.

Attending Surgeon to the Wills Eye Hospital; Fellow of the College of Physicians of Philadelphia; Member of the American and French Ophthalmological Societies.

THE original suggestion as to this character of work was kindly given to the writer by Dr. S. Weir Mitchell, of this city, in the fall of 1893, during a personal conversation upon various matters pertaining to physiological optics, colour-vision, &c. Dr. Mitchell desired to know why it was that artists could obtain more vivid impressions of the colours of a distant landscape whilst the head was lowered into a horizontal position. After hazarding, as a part explanation of the reason, that a greater blood supply was thus offered to the organ when the head was held in this way, and that there might be some difference in the combined colour values when refracted through another series of meridians of the dioptric apparatus, the writer concluded that a most interesting, and perhaps valuable, series of experiments upon the subject might be established amongst the abundant clinical material at hand, in one or more of his public services, and amongst his more intelligent private patients and his friends.

Limiting the present communication to those cases that gave full acuity of vision, had normal fields of vision, and showed healthy eye-grounds, a great number of observations were made by both the writer and several of his most competent friends and assistants.[1] In every instance, as far as practicable, the two series of experiments were performed under the same conditions. After the fields for white, yellow, blue,[2] red, and green were gotten in the ordinary manner, the patient or subject was placed upon a table or a sofa, so that the temporal or outer side of his eye was situated either superiorly or inferiorly. When in one of these positions, either before the perimeter or the gauged blackboard, and by both natural and artificial light, the same order of field-taking was instituted and consummated in various ways.

With the greatest precaution as to time of day, similar character of illumination for related experiments, and proper rest of the organ, the following observations were rendered sufficiently certain for publication.

(1) Equal areas of the same colour held at equi-distant points around the fixation object, when the fields of vision are taken in their ordinary positions, are more deeply saturated and more intense in vividity when they are situated at the temporal side of the point of fixation;[3] the relative positions of the order of brightness being temporally, inferiorly, nasally, and superiorly.

(2) Unequal areas of the same colour held at equidistant points around the fixation object show, when the fields are obtained in the ordinary manner, that the smallest colour areas are more distinctly visible and more deeply saturated to the temporal side of the point of fixation; the relative position of the necessary increase in colour area, to give the same vividity as that seen to the temporal side, being inferiorly, nasally, and superiorly.

(3) Equal areas of the same colour, studied during the method of ordinary field taking, are sooner perceived and

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[1] The writer here acknowledges his indebtedness for valuable help in this work to Drs. William Zentmayer, William Campbell-Posey, Frederick Krauss, Barton F. Potts, and Frank Savary Pearce.

[2] In many cases yellow and blue were omitted. In a large number of private subjects, however, yellow and blue were tried sufficiently often to admit of their entrance into the series.

[3] In some cases, the best point of excentric colour vision seemed slightly down and out from the point of macular fixation—*i.e.*, just beneath and between the fixation point and the physiological blind spot.

are brighter in tint when they are approached from the temporal periphery to the fixation object, than they are when they are brought in from any other peripheral border of the colour field; the order of greatest distance from the point of fixation in the different parts of the fields before the colours are recognised and properly named as such, being first, to the temporal side; secondly, to the inferior side; thirdly, to the nasal side; and fourthly, to the superior side.[1]

(4) Unequal areas of the same colour approached to the fixation point from the various peripheral borders of the fields of vision taken during the ordinary method of field-taking, show that the smallest colour areas are sooner perceived and become more quickly saturated, when they are brought in from the temporal side; the inferior and nasal sides being the next respectively and the superior side the last.

(5) In every instance where the fields of vision were obtained at right angles to the ordinary position, by placing the temporal field either superiorly or inferiorly, both equal and unequal areas of the same colour approached from the various peripheral borders of the fields of vision to the fixation point, show the same relative order of betterment of comparative sizes, positions, and saturations of tint, in that portion of the field which has been previously the temporal one.[2]

Conclusion.

As a logical deduction from these observations, and as a partial answer, at least, for the reason of the empirical method of dipping the head at right angles to its ordinary position, so frequently made use of by artists in order to obtain a better colour effect whilst observing a distant view, it seems most probable that the cause is, that by this plan, as it were, a stronger and a more highly evolutionised series of sensory elements are made to receive natural impressions which, under ordinary circumstances, are received and recorded less vividly by a series of lower grade and less powerful structures.

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[1] When care was taken to see that equivalent amounts of light were obtained in all meridians, the relative shapes of the fields of vision were not changed whilst the fields were tried at various angles.

[2] In fact, numerous experiments made since, which have no direct bearing upon the exact title of this paper, and which, with other important and interesting observations, will be reserved for a more extended communication, distinctly show that this is true, no matter how the temporal field may be placed.

A CASE OF SYMPATHETIC PAIN: PAIN IN FRONT OF THE CHEST INDUCED BY FRICTION OF THE FOREARM.

BY T. K. MONRO, M.B., M.S., GLASGOW.

THE patient, who was a fireman, aged 44, consulted me in the Spring of 1894, at the dispensary of the Royal Infirmary. On account of the curious nature of the case, I afterwards called at his house, traced out the important areas on the surface of the body with pen and ink, and then photographed him. The accompanying illustration is taken from the photograph.

The following are notes of the case (April, 1894):—Patient complains of pains in his chest and right arm. He is disposed to attribute them to a "cold" which he got three and a half months ago, while sweating profusely at his work. (He had no cold in the head, however; and, indeed, apart from pain in the chest, no symptoms of cold at all, so that his explanation is unsatisfactory, and it will be seen hereafter that injury was a more probable cause.) Excepting an attack of fever last summer, he never had a day's sickness till his present illness began. The pain came first into his right "shoulder," at the back. It began suddenly, whilst he was firing in the stokehole, and compelled him to stop work immediately. The ship's surgeon told him to lie on his back without turning. That pain has never left him since, though it varies in severity. It does not awake him at night, but it often prevents him from falling asleep. It is seated in an area (B—not seen in the figure) on the right side of the back of the chest, at about the level of the seventh dorsal spine. It is rectangular in shape, and nearly square, measuring an inch or two each way. The most painful spot is at the centre, where there can be felt what seems to be a localised thickening of the rib, as if there had been a fracture or other local injury. The continuous pain in this area B is variously described as a "dead burning heat," "like red-hot sand or iron passing through the flesh," or as if a knife were driving straight in. At first it extended up to the back of the head, but it does not do so now. It sometimes passed down to the small of the back, and then up again.

On the right side of the chest, in front, there is an area (F—marked out in the figure), bounded laterally by a vertical line extending from the axilla down to the sixth rib or thereabouts. The mesial boundary of this area is about a couple of inches nearer the middle line, and just external to the right nipple. Below the right nipple there is a spot which is sometimes painful (indicated in the figure by a cross). In the area F, "tearing pain" is felt. Occasionally, but only in consequence of exertion, *e.g.*, quick walking, or turning suddenly in bed, a momentary pain shoots across from this area towards the left side, about as far as the vertical line of the left nipple. (In the figure, the vertical line on the left front indicates the limit towards the left beyond which this shooting pain does not pass; the horizontal line shows its usual level and direction.) The momentary shooting pain is generally followed by a feeling of "pins and needles," lasting for five or ten minutes, at a point just below the right nipple.

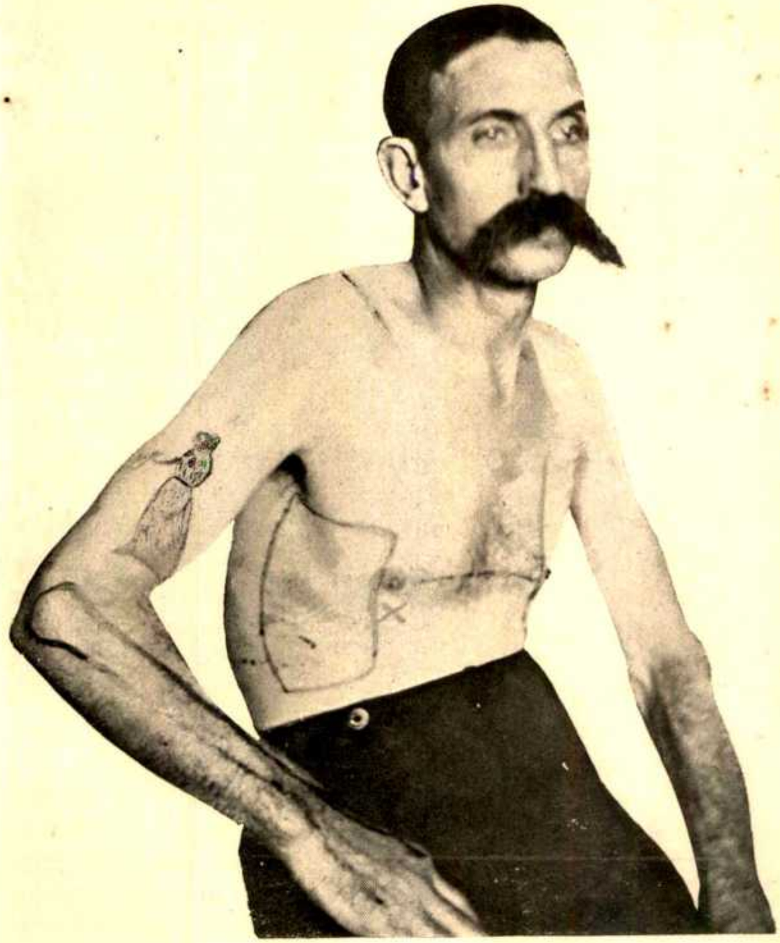
A third painful area to be described (A) is on the posterior aspect of the right forearm (shown in the figure). Pain here is specially apt to be induced by pressure over the radio-humeral articulation. The area of pain extends slightly higher than this, and includes the middle two (longitudinal) fourths of the dorsal aspect of the forearm; it reaches as far as the four inner metacarpo-phalangeal articulations. The five digits, however, are free from pain. The surface A is about an inch broad at the middle of the forearm, which is itself two and a half inches in breadth at this part. A narrows at the wrist and widens again towards the bases of the fingers. When pain is induced at the elbow-joint, it is always severe; it may be associated with slighter, but occasionally with quite as severe pain further down the limb. About a month ago, the pain in the right elbow was so severe that the limb was practically useless. Bending the elbow causes severe "cutting" pain just above the external condyle. Flexing the wrist also causes a slight, but not nearly so great pain at the elbow. The joints are never swollen.

One of the most remarkable features of the case comes now. *A touch on any part of A* (but not on any other part of the forearm) *is felt in F* (chiefly at its middle part), *in addition to being felt in A.* The converse is not the case. *Firm pressure on A* (if the region of the elbow be avoided) *causes no pain in A, but causes severe "tearing" pain in F* (chiefly at its middle portion).

F is the only part of the trunk in which pain is induced by handling the arm. Rubbing the dorsum of the forearm induces the pain in the chest; indeed, the patient seems to have first discovered this sympathetic pain on one occasion when he was washing his arms, about a month after the accident to be presently described. He says it is much worse when he uses cold than when he uses warm water for washing.

Extension of the right wrist causes no pain at the elbow, but flexion of the fingers causes so much pain as to prevent him from gripping objects with his right hand. There appears to be no tenderness of the nerve-trunks in the forearm.

There are pains occasionally, but only for a second at a time, in the knees and ankles; these pains very seldom occur in the left knee, and are not so severe there as in the right knee. Apart from this, the left leg is perfectly well, and the left arm and left side of the trunk are healthy. There is no weakness of the legs; the ground feels naturally to the feet; the knee-jerks and the pupils are normal. There is no nystagmus, ataxy, headache, or vomiting. The bowels are regular. There is no *tache cérébrale.* The heart and lungs seem healthy, and there is no evidence of



DESCRIPTION OF FIGURE.

The area A is seen marked out by ink on the back of the right forearm and hand. A tattooed figure is seen on the upper arm. The area F is marked out on the right side of the chest, and a cross below the right nipple indicates one seat of pain. Friction or simple pressure over A causes pain in F.

aneurism. The pectoral muscle-fibres are more irritable when struck on the right than on the left side. There is no obvious atrophy of the muscles in the right hand. Patient says he has never had rheumatism. He has had no cough or spit in the course of his illness, but he breathes cautiously, because a deep breath is apt to catch him in the right side.

His chest was extensively blistered by a doctor about four months ago. The blistered surface healed within a few weeks. One doctor told him he had "pleurisy," another said "dry pleurisy," and a third said he had "pleurodynia."

Careful inquiry reveals facts that assist greatly in explaining these curious symptoms. About eight days before patient became ill, he stepped upon an insecure plate in the engine room, and lost his footing. The result was that he struck his elbow (? A) against something which caused his elbow or fist to strike against his chest in front (? F); after which his body was twisted round so that his back (? B) knocked against a corner. He subsequently felt some soreness about B, but he did not think—at the time, at least—that anything was broken.

Patient states that for years, *i.e.*, since long before his illness began, he has sweated greatly, and particularly in bed, with his right side only. This side, as he has noticed hundreds of times, will be soaking when the left is dry. He may find his right ear filled with perspiration. The right side perspires in this way all over, but he says that both sides sweat alike when he is at work.

When a patient complains that rubbing his arm causes pain in his side, it is perhaps natural that the idea of allochiria, as described by Professor Obersteiner, or of allachæsthesia, as described by Sir Grainger Stewart, should occur to the mind. This case, however, does not conform strictly to either of these types as originally portrayed. At the same time, it must be remembered, different writers on allochiria have shown that one particular form of sensation (touch, pain, temperature, tickling, sight, hearing) may be affected by itself. If the present case is allied to allachæsthesia (rather than to allochiria, where the sensation is referred to the opposite side of the body), pain is the form of sensation involved; for pressure or friction on the forearm, which is not such as to cause pain locally, causes it to be felt in front of the chest.

On the more convenient hypothesis that the case is an example of sympathetic neuralgia, the nerve-connexions between the two parts become interesting and important. In normal individuals, the area A in the forearm is supplied by the lower external cutaneous branch of the musculo-spiral nerve and by the posterior branch of the musculo-cutaneous. As Ross has pointed out, the cutaneous nerve-supply of this region is homologous with that furnished in the trunk by the dorsal portion of each anterior primary division of a segmental spinal nerve, including, therefore, the lateral cutaneous nerves of the thorax. The figure shows that the area F on the surface of the chest must be almost entirely supplied by these lateral cutaneous branches. As the forearm, however, is innervated from the cervical cord, and the particular part of the chest-wall in question from the dorsal cord, at a considerably lower level, the special path of connexion between the chest and the forearm in this case is not very easy to comprehend.

Reviews and Abstracts.

Entwurf zu einer physiologischen Erklärung der psychischen Erscheinungen. Von Dr. SIGMUND EXNER, o.o. Professor der Physiologie und wirkl. Mitglied der kais, Akademie der Wissenschaften zu Wien. I. Theil. Mit 63 Abbildungen. Leipzig und Wien. F. Deuticke. 1894. Pp. viii., 380.

THE "explanation" of a scientific phenomenon consists in the statement of the conditions under which it occurs. The psychologist explains a given mental process by analysing it into its simplest conscious constituents, and by referring these to their physiological substrate. An enumeration of the physiological conditions of states of mind is held to be essential by modern psychology: Wundt's conception of "physiological psychology" is practically the same with Fechner's idea of "internal psychophysics."

It is in this, the physiological sense, that the word "explanation" is employed by Professor Exner. "By an explanation of psychical phenomena I mean their reference to physiological processes in the central nervous system already familiar to us in other connections." "The problem which I have set myself is that of the reference of the most important psychical phenomena to differences of degree in the excitatory condition of nerves and nerve-centres, *i.e.*, the reduction of all the [qualitative] variety of consciousness to terms of quantity, and to the diversity in the central connections of nerves and centres which in all other respects are practically undifferentiated." If the work arouses distrust at first sight by the number and prominence of "unproven, hypothetical propositions," we must remember that principle, rather than detail, is at stake. It is enough to have shown that mental processes are capable of scientific treatment; particular hypotheses may be looked upon with comparative indifference. At the same time, the author realises the necessity of basing hypothesis, wherever possible, upon a foundation of ascertained fact, and brings to his task of explanation the interest and experience of a quarter of a century.

The book is not written for the general reader, but for those who have mastered the elements of physiology or psychology. To students of the latter science it makes a very special appeal. They are accustomed always to regard the psychical as primary, and the physiological as secondary: their analysis begins with the mental. Professor Exner reverses this order, and passes from the nerve process to the correlated conscious process. Psychology, in his series of chapters, takes on a new appearance: he begins with voluntary movement, and goes on to deal successively with attention, sensation, perception, idea, and the phenomena of intelligence. A second Part is to discuss more special topics—the relations of the separate divisions of the central nervous system to the various mental functions. It is not to be expected for some years to come.

Chapter I. is a preliminary exposition of the anatomy of the central nervous system. It is intended to assist psychological readers in their understanding of the book, and is well adapted to its purpose. Though very greatly condensed, it is clear and simple in statement, and covers the most important facts in an adequate way.

Chapter II. gives the physiological data and inferences (*die physiologischen Grundphänomene*) upon which subsequent theories are based. The author says modestly that it "contains nothing new." But it has the great value—apart from its function as the foundation of the whole work—of being the first attempt at a general physiology of the central nervous system. It is undoubtedly coloured to some extent by the thought of the purpose to which it is to be put; but departures from established fact are frankly termed hypothetical, and nowhere involve any contradiction of physiological laws. The first of the five sections deals with the conduction of excitation within the nerve. The second discusses the central "exchange" (*Umsatz*), "the transference of a stimulus in the grey matter . . . from one known path to another path, of which the existence at least is known," under the headings of reflex movement (reflex time, summation of stimuli, the "explosive" process in the reflex, theory of conduction in the grey matter, the grey nuclei) and concomitant sensations, cases of the "transference of an excitation from one centripetal path to another." We may notice here that the ganglion-cell is spoken of as "charged" and "discharged"; that fibre masses or ganglion-cells are "related" according to the closeness of their interconnection, anatomical or physiological; and that the cerebral cortex receives the name "organ of consciousness." The third treats of central inhibition (of motor and sensory impulses) and facilitation (*Bahnung*). The latter concept seems destined to play a large part in physiology and psychophysics: it is interesting to compare Professor Exner's illustrations with von Kries' paper upon predisposition (*Einstellung*) in the *Zeitschr. f. Psychol.* (vol. viii., pp. 1 ff.). The two last are entitled "Interaction of Excitation in the Grey Matter" (tetanic reflex action, the physiological identity of the mechanism for tetanic reflex action and for leaping, combination of successive movements) and "Sensomobility," the "capacity of man or animal for movement, influenced, controlled, or conditioned by centripetal nervous excitations" (the true reflex, characterised by subcortical regulation; instinctive movement, regulated by intention—*Intentionsregulirung*—in which the attention effects the temporary installation of a subcortical reflex apparatus, subserving the intended purpose; and conscious movement, with cortical regulation). The processes involved are exemplified by elaborate anatomical-physiological diagrams. The rubrics of the five sections will suffice to give the reader some idea of the terms in which the author has couched his psychophysical explanations. The hundred pages of the chapter hardly admit of any other kind of summary, occupied as they are either with progressive argumentation or with detailed evidence upon particular points.

The transition from sensomobility to voluntary movement (Chapter III.) is an easy one, just because the writer is concerned with the physiological side of the process, and can neglect its psychological *differentia*. "The fact which stamps these actions as voluntary, the fact, *i.e.*, that they are the result of impulses of the will, and as such are performed with consciousness," need not be considered for the time being. Beginning with combinations of voluntary movements, Professor Exner disclaims the view that voluntary motor impulses are directed upon the innervation of a particular muscle group. It is the object, the result of the movement, which is in consciousness. The result is given in terms of sensation; and if sense is impaired, the movement becomes difficult or impossible. Voluntary movements are of two kinds, mixed and pure. The act of swallowing is taken as an illustration of the former, speech as an instance of the latter. Most of the "voluntary" acts of animals are mixed: cortically initiated, but thenceforward regulated by subcortical centres. In man, subcortical regulation has not disappeared, although it is largely subordinate to cortical. Indeed, the cortex is probably operative in many cases of "unconscious" action, as, *e.g.*, in the innervation of trunk and legs which accompanies the conscious movement of the arm when an axe is lifted to strike,—witness the effect of practice and attention. On the other hand, subcortical influences are to be traced in pure voluntary movements (innervation of unnecessary muscles, symmetrical concomitant movements).[1] The difficulty of executing a voluntary movement for the first time—*e.g.*, when a child is learning to talk—consists "in discovering and hitting the right cortical paths, where there are plainly so many which are closely related to one another." Innervation and inhibition probably co-operate to produce the intended result. In the illustration chosen, the intended result is the production of a simple sound. The mechanism of this sensory regulation, or regulation by idea, differs only in the range of its possible effects from that of the regulation of the instinctive movement "by intention." The child, in the course of performance of numberless voluntary movements, comes by chance or by imitation of mouth-movement to say the particular word. An association is set up in the cortex between the sound and the voluntary impulses leading to it. Repetition strengthens the association, and finally brings knowledge of the muscular innervation involved in the production of the sound. But the cortical association means a relation between the corresponding subcortical centres: repetition of the sound, *i.e.*, simultaneous excitation of the paths for hearing and speaking, sets up an "intercellular tetanus" in these centres. Now, therefore, the idea of the sound, excitation from the sensory end, begins to be important for correct innervation; for "attention to the sense impression" or the "idea" of it acts by way of facilitation upon subcortical centres. The theory is supported by the empirical observation that no movement whose sensory effect cannot be ideated is under the domination of the will.

The following section, on the temporal course of voluntary movements, analyses the physiological process in the reaction experiment, simple and compound. The phenomena of facilitation, and the influence of idea on voluntary movement, are visible in the introspective and objective results. The paragraphs are timely, as a contribution to the theory of the reaction, apart from their special bearing upon the matter in hand.

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[1] The subcortical centres for instinctive movement (regulation by intention) are probably the thalami and corpora quadrigemina; those for voluntary movement, the caudate and lenticular nuclei (Meynert, Nothnagel, Bechterew).

The author passes in Chapter IV. to the consideration of the attention, which has been mentioned before in connection with instinctive movement, and examined under certain aspects in the discussion of the reaction experiment. "We have seen that the act of will produces changes in the nervous system, the effect of which is, that an excitation now follows a path A, and now, when a different change has been voluntarily set up in place of the former one, takes the path B. Changes thus set up have the character of states (*Zustande*). I regard this interaction of different parts of the central nervous system as the essential thing in what we call attention (*Wesen der Aufmerksamkeit*)." The attention, however, is not simply concerned with the interaction of sensory excitation and motor direction; it may be exclusively sensory, though, it is true, there always remains a tendency to movement. In any case, "whether it is turned to sense-impression or movement or memorial image, the attention has a two-fold function: it makes certain paths within the nervous system especially practicable, and holds them open for some time, more or less; and it reduces the conductive capacity of a large number of other paths." This state of the system is termed *Attention* (not *Aufmerksamkeit*). It plainly consists in an "attentional facilitation" and an "attentional inhibition." The latter implies "an increased tonus of the cells, in spite of which their discharge is rendered difficult." The former might be thought to imply an enhancement of the sensation attended to; but Professor Exner shows, by one of his characteristic schemata, that the conscious result of stimulus-increase is different from that of attention-increase. Sense-impressions have been "worked over" at least once (in the ganglia of the caudex cerebri and sensory nuclei) before their impulses are affected by attentional facilitation and inhibition. There are certain centres to which the attention has no access (those for cardiac movement, for the pupillar reflex, &c.). It can influence the results of all movements and inhibitions, the execution of which may be accompanied by consciousness.

This account of the physiological equivalent of attention evidently demands a mixed, positive and negative, psychological theory of the state. Neither a purely positive explanation, like

G. E. Müller's assumption of a "centrosensory reinforcement" of peripheral excitations, nor a purely negative one, like Külpe's "inhibition" hypothesis, is adequate to the facts of nervous function.

The remaining four chapters of the work take up the examination of the contents of consciousness, in the order from simple to complex. Chapter V. ("Sensation") opens with a statement of the general law of the physiology of sense:—"Every sensory nerve fibre, however excited, brings a sensation to consciousness, which differs from any sensation that another nerve fibre can mediate." In order to distinguish this single-fibre sensation from the "sensation" in the sense of a presentation complex, it is termed a "sensation element." Even the sensation element has a comparatively complicated physiological substrate, as is shown by the fact that it possesses quality or modality (in virtue of which it is comparable with other sensations), a variable intensity, and, in certain cases, a local sign. Variation of intensity involves change of quality.[1] The writer rejects Helmholtz' definition of sensation in favour of its characterisation as "an unanalysable portion of a sense-impression, with no attributes beyond those of quality and intensity." "The description of a sensation is unintelligible except for those who have it in memory, if not for the moment in consciousness."

Where the law of isolated conduction from sensory nerve termination to cerebral cortex is valid, we may speak of "primary" sensations. But it is probable that the range of its validity is much smaller than is usually thought—if, indeed, it is really valid at all. Beside the primary sensations, then, we have "secondary," which "arise from the interaction of two or more excitations in nervous organs, simultaneous or successive." There is no conscious mark of secondariness. Secondary sensations are local (a hunter can just see the game in the twilight, it becomes invisible when he closes one eye to take aim); temporal (tickling of the lips in playing a trumpet); and both (sensations aroused by the movement of a perceived object). "Change as such excites a specific sensation."

The feelings are "sensations from internal organs, arising secondarily as the result partly of centripetal, partly of centrifugal excitations, and arriving at the organ of consciousness as do other sensations," *i.e.*, are a kind of concomitant sensations. They are ordinarily set up by "the release by a central or peripheral excitation of a subcortical central process, which either evokes muscle sensations directly or starts centrifugal stimuli, which travel to the muscles and cause the sensations of muscular contraction in the corresponding sensory nerves." The most important feelings are pleasantness and unpleasantness, each of which is independent, *e.g.*, of the degree of intensity of other simultaneously present sensations. They consist essentially (1) of sensations in the thoracic cavity, due to changes of circulation, and (2) of "muscle feelings." Mixed feelings occur, in consequence of the partial identity of the muscle groups concerned. Some nerve endings always respond to excitation by pleasure, others by pain. The. pain following intensive excitation of a "sensory" nerve, in the ordinary sense, may be mediated by special pain fibres, or by the "sensory" fibres themselves. A diagram shows the functions and connections of the "pain centre," which is regarded not as a separate central organ, but as composed of "the higher nervous centres of all the muscle groups which are in any way concerned with the movements of defence. These may be situated in quite different parts of the central nervous system."

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[1] This statement rests upon an objection to Fechner's law, urged in a different connection by Hering. It is that, if the law holds, "the increase of a 1cm. line to 10cm. must cause a sensation indistinguishable from that caused by the increase of a 1m. line to 10m., *i.e.*, 9cm. must appear to us as large as 9m." This is surely an erroneous interpretation.

A concluding section treats of "combinations of movements and secondary sensations in their relation to inheritance." The author shows how the reflex may be derived from the voluntary action, and how the implied cell-relationship may be inherited, on either of the current theories of hereditary transmission. There is a continuous series from strictly secondary sensations to analysable complexes (violin clang); and from irresolvable combinations of movement to combinations which can be broken up. The mechanism of the less rigorous connections is given in diagrammatical form.

A perception (Chapter VI.) is "a unitary complex of excitations, which can be split up by consciousness into sensations." There is no absolute line of distinction between sensation and perception; the passage is by way of the analysable secondary sensations. Perception becomes idea by association with memorial images. This complex, which Helmholtz, *e.g.*, technically names "perception," Professor Exner calls "secondary perception," reserving the term "primary perception" for the perception of his original definition. The chapter falls into three sections. (1) *The excitations in the organ of consciousness.*—The cortex is composed of nerve paths, in the widest sense of the phrase. It is a result of physiological thinking, to be verified *à posteriori*, that "all the qualitative and quantitative phenomena of conscious sensations, perceptions and ideas, are referable to quantitatively variable excitations of different portions of this sum of paths." Two things follow. "Two sensations are identical for consciousness, when the sense-stimulus excites the same cortical paths to the same extent." And "two sensations are similar, when at least a portion of the paths excited in the two cases are identical." Since the path of a sensation element divides at the centre, or between periphery and centre, "the quality of a sensation and its local sign are the result of the excitations of different paths within the cortex." These four propositions are fundamental. It may be inferred from the last that there is a "perception element," the sum of sensations due to the excitation of a single sensory fibre at the periphery; an element, *i.e.*, which has a highly complex physiological substrate. Not all the cortical excitations of the moment are represented in consciousness, but all may influence conscious content, and may be consciously apprehended (thus differing from the excitations underlying strictly secondary sensations). "These cortical excitations, which do not come independently to consciousness, play a large part in the psychical life." (The reader is reminded of Muller and Schumann's work on "Memory," or Kulpe's on "Attention.") The phenomena illustrate a principle already employed by the writer in other connections, that of "central confluence." "A sum of excitations in the cortex, following different paths, can act upon consciousness as a single excitatory process." The cortex is never wholly unexcited. (2) *The Primary Perceptions.*—Like sensations, these are really abstractions. The primary perception "is the process which we must suppose to be present in the cortex of a newly-born child, or of a congenitally blind person, deprived also of tactual and muscular sensation, who has suddenly been given the power to see and feel." (3) *The Secondary Perceptions.*—Here we have to consider, in the first place, the "local impressions" which constitute, *e.g.*, the "consciousness of direction," so highly developed in many animals and some men. Ruling out visual sensations for the moment, we can analyse this consciousness into "obscure" perceptions and recollections (innervation and organic feelings, recollections of obscure perceptions of bodily attitude). "Processes run their course in the cortex as obscure perceptions, and their results are embodied in consciousness, without its being possible for the factors which enter into them to be apprehended by consciousness after their occurrence." Local impressions lead us, secondly, to the "principle of recognition," of our capacity of distinguishing whether a cortical excitatory process is new or repeats an earlier one. Our recognition of the excitation of a path is explicable in terms of two physiological laws. The first is that of the "rutting a path" by usage—the setting up of a kind of permanent facilitation. The other is that of the arousal of secondary excitations. A bloody finger means the excitation of the rutted red-path. But this path was traversed previously by the redness-excitation of a cherry, and an "intercellular tetanus" effected between all the cherry-cells. The blood consequently excites (weakly) all the cherry-paths, by exciting (strongly) the red-path. These excitation complexes, which do not correspond to the condition of the subcortical centres (being due to attentional facilitation), are "memorial images." It is to be noted that a cortical excitation complex need not be for consciousness the sum of the separate excitations, though it will resemble that sum. (We may *cf.* Kulpe's definition of sensation fusion.) Thirdly, we come to the eye, to the retinal "local signs." There are two constituents in local signature. "Muscle feelings" account for the "sensation complex, evoked by a visible object as a whole." To explain "how parts of an object or several different objects can be correctly cognised as regards their relative position with apparent simultaneity," it is necessary to call in the aid of "unitary secondary sensations, varying with the intensity of excitation of the particular paths which belong to them." Subcortical relations are developed between the central elements connected with neighbouring retinal elements, or with corresponding elements of the two retinæ. We must also assume a cortical relationship between the terminations of "similar," *i.e.*, neighbouring retinal elements. The discussion of the two factors in local signature is very elaborate. Reference is made to the development of the muscle feelings in the animal kingdom under the general law of teleology; and their action is demonstrated by a diagram, like that used to illustrate the visual "movement sensations" of Chapter V., 2. Another diagram shows the cortical network corresponding to the relations of the retinal elements. Pathological cases are adduced to support the theory that localisation occupies different parts of the cortex from those concerned in the other functions of vision. Localisation may be impaired alone; vision may be practically abolished, with the exception of the movement sensations; colour sensations may be absent, while those of brightness and locality are retained.

The "similarity" of sensations is examined, in the light of the law of specific function (Chapter V.). Lastly, the complication of the perceptions already spoken of by the perception of depth is indicated.

Chapter VII. treats of ideas, which differ from perceptions in two points. The idea is a perception *minus* certain excitations in the cortex—those which represent sensation in its purest form, as least "worked over" psychically, *i.e.*, those of the radiation of the sensory nerves. And the result of attentional facilitation differs in the two cases; in perception, it finds excitation in the subcortical centres, in idea it finds them at rest. Individual variations are more important in idea than in perception. The underlying excitation is diffused over a large portion of the cortex. There are "obscure ideas" as there are obscure perceptions, playing their part "outside or almost outside of consciousness."

The first section of the chapter raises the question of the nature of this (1) *consciousness*, of which so much has been said. "When an excitation-complex in my cortex has attained a certain diffusion . . . and has so come to involve the paths which have been more particularly excited by events of my own experience . . . and which are therefore almost always in a state of facilitation—the paths, in a word, which belong to my idea of myself; and when the excitation has increased in intensity by way of intercellular tetanus, owing to the number and variety of the excited fibres, and the excitation-complex has consequently acquired the . . . peculiarity of inhibiting weaker excitations; then I say that the idea is in consciousness." The consciousness "into" which the idea "enters" is a group of associated ideas. Self-consciousness "depends exclusively upon ideas of previous experiences, more or less distinctly associated to the ideas or perceptions present at the moment." The cortical fibres concerned in voluntary action (Chapter III.) form part of the complex whose excitation gives the idea of self. (2) *Ideas in their Relations to Voluntary Movements.*—Before acting, we have an idea, however transient, of the result of our acting. Having acted, we possess a perception, with which we can compare and by which we can check our idea. The mechanism of these processes is explicated. (3) *Ideas in their Relations to Sensations and Perceptions.*—Very different primary sensations can produce practically identical perceptions, because of the wealth of the cortex in associative "ideational material." On the other hand, this ideational material can facilitate or inhibit sensations. The modificatory processes are probably cortical, as well as subcortical. Instances are given under both rubrics. (4) *The Interrelations of Ideas.*—In the last resort, all ideas originate in perceptions. We are able, however, to bring about relationships between cortical paths which have not been previously effected by perception. The limit of imagination is reached when the path-complexes excited are partly identical: we cannot ideate a "curved straight line" (identity of the motor mechanism). No idea can be retained for any great length of time. It fluctuates intensively, probably for vasomotor reasons. It also oscillates with other ideas, probably as the result of the rapid exhaustion of the cortical organs. (5) *The Arousal of Ideas by Words, and the General Effect of Sensational Succession.*—The fact that the spoken words "St. Paul's" arouse the total idea of the cathedral can only be explained by the consideration that these words constitute a succession of sounds. Other words contain similar separate sounds ("pall," "awe"); but as the two are uttered, there is a progressive restriction and definitising of the cortical paths excited. And this holds of single words. The most primitive languages employ sounds as such; the more developed, sound-successions. Although we have in adult life no consciousness of understanding words which we hear in terms of their sequence of letters, this is only because of the extreme rapidity of the process of "successive selection." Sequence-understanding is rendered possible by the persistence of nervous excitations after the cessation of stimulus.[1] It is true that the motor innervations of the listener play some part in his understanding, but they cannot be the sole factor. The idea of the word "St. Paul's" is, of course, different from the idea of "St. Paul's." It has visual, motor and auditory constituents. The two ideas are closely associated. The auditory word idea is of brief duration, and can only be revived by aid of the motor.

We have now reached (Chapter VIII.) the most complex mental processes—the phenomena of intelligence. (1) *Forms of Thought.*—The formation of a *judgment* can take place in two ways: we judge, when a difference between ideas, given as different, is apprehended (*erfasst*) by consciousness; or when we analyse out, by attention, the constituent parts of the excitation-complex corresponding to a single idea. If we direct consciousness upon the cortical processes common to a number of ideas, we have a *concept*. Judgment and concept are thus not necessarily connected with language, though language is undoubtedly of service for both—more important for the concept than for the judgment. *Inference* (*cf.* J. S. Mill and Helmholtz) is a "form of words, in which is expressed a judgment together with the circumstances which led to its formation." *Reflection* always involves "an intensive attentional excitation of all the cortical fibres constituting the idea which is the object of reflection, and the consequent facilitation of related fibres, near and remote." This given, thought may follow either of two roads. We may have a "happy thought"—a new association being formed between ideas hitherto separated—or we may "experiment" by facilitating all manner of related paths, and looking out for contradictions in the excited ideas. Serious thinking ordinarily depends upon a combination of these processes. A *resolution* is a judgment passed after experimental reflection, and is always on the side of the ideas which are associated with the maximum of pleasure or the minimum of unpleasantness. These feelings are represented in the cortex directly, but also indirectly (reaction from sub-cortical centres). It must be remembered that the part played by the will in the processes of reflective thought and resolve need be but very small. "Thought goes on in me" is a truer expression of the facts than the customary "I think." (2) *Instinctive Feelings and Thought.*—It is more than possible that "a connection of a cortical complex of a definite kind with the centrifugal fibres running to the feeling centres will ultimately take its place among the transmissible nerve paths, provided that it has subserved an important physiological function for many thousands of years." Ideas, it is true, are not inherited; but there may be transmitted a disposition, an inclination towards the colligation of certain not sharply defined ideational masses with determinate feelings. The feelings are "measures of defence" taken for us by nature. The associations of feeling and idea of which we are speaking result in the protection of the individual, the benefit of his immediate descendants, or the benefit of society. The organic faculty of unconscious action for a definite end we call "instinct." "An experience which runs counter to our instincts is painful, and there is no other means of causing pain." Hence the excitations in the centres for pleasure and unpleasantness may be termed "instinctive feelings." These feelings are discussed at length under the three headings just mentioned—their discussion taking the author some distance into the domain of ethics and sociology.

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[1] It must be remembered that the memorial images of the separate sensations differ not only in intensity (according to their temporal order), but also in quality (*cf.* Chapter V.).

(3) *Causal Thinking and Free Will.*—"There are congenital relationships in the human cortex, as a consequence of which the sensation of a change (which, of itself, attracts the attention, and, coming to consciousness as perception, affects a portion of the motor cortical area) excites further, and more especially motor paths subserving the search and effort required for the investigation of the change, *i.e.*, the obtaining of associations for it. So far as regards the change, the only associations that can be found are 'causes.' This congenital endeavour after the associations of the change we term the law of causal thinking." Since ideas are not inherited, it is an inclination only, not a constraint, which is congenital. There are biological reasons for the importance of the perception of change. It is curious that although we cannot conceive of effect without cause, most of us actually recognise uncaused effects. While we are dealing with the external world, it is plain sailing with the law of causation. The objective psychology of the present book, *e.g.*, is causal. When we speak as experiencing selves, however, we cannot run the causal chain further back than consciousness; we know nothing of the unconscious physiological processes posited by the man of science. Hence, to the naive mind, "free will" is the last cause of action, the uncaused effect with which the causal nexus begins.

Two remarks seem called for by way of corollaries. (*a*) We cannot clearly ideate a process without cause. Hence the causal law is a psychical phenomenon. But we think that we act by free will; this is also a psychical phenomenon. We need not strive to reconcile the contradiction. Causation may be the most useful guide in one sphere (scientific thought); free will in another (individual and social education). That the causal law is built into the physical structure of man, in the manner described, makes no difference to this conclusion. (*b*) We have spoken of free will; we have not yet said what "will" is (cf. Chapter III.). It is nothing *sui generis*. When the cortical cells are sufficiently charged, *e.g.*, after sleep, some stimulus excites a group of paths, and intercellular tetanus—the play of ideas—begins, whether we will or no. "There commences the normal interplay of associations and (where the excitations are transferred to motor cortical fibres) of actions, which are performed in accordance with the associations, and present themselves to the subject as voluntary actions." Their energy is due to the charge of the cells (sleep) and to all the associative excitations in the cortex (also intensified by sleep). At the close of the day, the "energy of the will" is lowered; action becomes weaker.

The index is scanty, and not always correct.

There can be no doubt that Professor Exner has worked out a plausible and consistent theory. This is not to be wondered at, when we remember that the *Entwurf* is the result (as I have said at the outset of the present notice) of twenty-five years' occupation with physiology, "during which the attempt at an explanation of psychical phenomena came to be more and more definitely regarded as a life-work." The greater part of the author's published writings sprang, as he tells us, from this one interest. I have aimed, in the above summary, at bringing out the psychology of the volume, that the reader may be able to compare the conclusions reached by purely physiological methods with the outcome of strictly psychological analysis as given in the best modern text-books. The summary is so brief that very many points of detail, important in themselves, have been passed over; while it is of the nature of a summary to emphasise the breaks in the argument rather than its continuity. Moreover, it has been impossible even to mention the light-giving personal experiences with which the discussion is interspersed. And nothing has been said, beyond the barest statement of their existence, of the nervous schemata which form a large feature of the work, and constitute an integral part of the writer's "explanation."

As a constructive synopsis of the scattered results of physiological investigation of the central nervous system, and as the first thoroughgoing attempt at the realisation of an "internal psychophysics,"[1] the *Entwurf* will be extremely valuable both to physiologists and psychologists. Under either aspect, of course, the book has the defects of its qualities. We see the weak places in the methodological framework which holds the physiological data together; and we see, I think, that current physiological concepts are incapable of furnishing a really adequate "explanation" of mentality. That, at least, is the impression left by the work regarded as a whole; and the impression has been confirmed by a detailed comparison of psychical facts and alleged physiological conditions in two or three special cases chosen somewhat at random. Professor von Kries has worded this judgment more forcibly from the physiological point of view than I should have ventured to do from the psychological. "I cannot get rid of the thought," he says, "that psychology presents a whole series of problems for which current physiological ideas do not furnish any adequate expression. . . . These considerations have kept me in doubt as to whether the physiology of the central nervous system, and more especially of the cerebral cortex, must not come in the future to work with quite different ideas from those which it now employs (excitatory process and its conduction)."

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[1] This characterisation may seem to conflict with what was said above of Wundt and "physiological psychology." But a comparison of the *Grundzüge* with the *Entwurf* will show what is meant. Wundt devotes 150 pp. and three chapters to anatomy ("structural elements of the nervous system," "morphological development of the nerve centres," and "course of the paths of nervous conduction"): Exner 80 pp. only. Wundt's two physiological chapters ("physiological functions of the central parts," "physiological mechanics of the nervous substance") and Exner's one (*die physiologischen Grundphänomene*) are of approximately the same length; but what in Wundt is a summary to be referred to for "explanation" in later chapters, is in Exner introductory to an elaborate series of very detailed explanations, which practically constitute a continuation of it. The problem is essentially the same; the way of approaching the problem is different. Wundt connects a minutely subdivided mind with a generalised substrate; Exner a minutely analysed substrate with a somewhat roughhewn mind. It is the definitising of the excitation-process, however, which is the keynote of "internal psychophysics."

E. B. T.

Various Forms of Hysterical, or Functional, Paralysis. By H. CHARLTON BASTIAN, M.A., M.D., F.R.S. Lewis, London, 1893. 1 vol., 8vo., pp. 197.

DR. BASTIAN'S work is well timed and of great interest. He applies to the study of hysteria a method formerly almost exclusively employed, but of late years—perhaps erroneously—somewhat neglected. We mean the *anatomo-pathological* method, which has given such brilliant results in the study of nervous diseases, and which had been also attempted in the interpretation of hysterical phenomena. Thus our respected and regretted master, Professor Charcot, compared,[1] in his early work, hysterical hemi-anæsthesia with that observed in brain lesions, especially in those of the posterior part of the internal capsule.[2] Dr. Paul Richer did the same in a memoir (1882) recently published in a work on hysterical palsies and contractures (1892), when he described symptoms due to disordered cerebral innervation, and paralyses, "the dynamic lesion of which must be sought in the inferior part of the cerebro-spinal axis." Bastian thinks that this method has not yet given all its promised results, and that it must be applied again and more rigorously. His book is an interesting and enterprising effort: it is an anatomical study of hysteria.

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[1] Charcot, "Leçons sur les Maladies du Système Nerveux," i., 306.

[2] *Ibid.*, iv., 114.

Anatomo-pathological studies are usually done by dissection, microscopical examination of organs, and minute comparison of observed lesions with symptoms observed during life. Unfortunately, this procedure, rarely applicable in hysteria, has hitherto failed to yield the least result in the study of the disease; and on this point the work before us does not differ from the others. Though an anatomical book, it does not contain a single figure. The author is obliged to have resort to the indirect and much more dangerous method, already employed by Charcot—the method of *comparison*. Hysterical symptoms, with unknown physical substratum, are compared with those of other more or less analogous diseases, but with known lesions, and conclusions are drawn from the ones to the others. Thus contractures, or some contractures, in hysteria are assimilated with hemiplegic spasms through descending sclerosis of the pyramidal tract; some hysterical paralyses are compared with atrophic infantile palsy through anterior cornual lesions; and one concludes that the hysterical contracture depends upon a lesion of the pyramidal tract, the paralysis upon a lesion of the anterior horns. This mode of argument, which pervades the whole book, arises from the author's conception of the so-called functional diseases as compared with the organic, or lesional, diseases. We must examine this point more closely.

Neuroses, such as hysteria, of which the lesions escape our eyes, are often considered as functional or dynamic disorders, as opposed to the organic disorders with known lesional substratum. These very vague expressions merely serve to hide our ignorance. Bastian very rightly tries to give precision to this idea. He does not look upon the two as so distinct as is usually done; and he shows well how in some cases a diagnosis is difficult, and that hysteria can show incontinence of urine, atrophy, even eschars (very rarely, we think), which had been considered as characteristic of organic diseases (pp. 85, 117, 123). He insists, with several recent writers, upon the frequent association of hysteria with organic lesions, such as insular sclerosis—Pott's disease (pp. 67, 72). He even thinks that in some cases a long continued functional paralysis may be transformed into one with actual lesions. Bastian recalls an old saying of Charcot's,[1] that a long continued hysterical contracture may bring about a sclerosis of the corresponding pyramidal tract. We think, how-

[1] *Ibid.*, i., 362.

ever, that as a natural consequence of his prolonged researches, Charcot would not have latterly accepted, without reserve, this opinion; and it is a pity that our author should not have fortified this old view by fresh proofs. Bastian concludes that there is a great analogy between the two forms of nervous diseases, which really differ in three points only: (1) Lesions have not been observed in functional disorders; (2) Their symptoms are merely attenuated, reduced. "Looking at the matter from another and, I venture to think, more rational point of view, we may see that the phenomena of mere functional disease should not be expected to differ in kind, but merely in degree, from the forms of structural disease to which they are akin" (p. 122). (3) Functional diseases are chiefly characterised by their lesser duration, by their recovery. In a word, the lesions in functional troubles are slight, transitory, due probably to some vaso-motor spasm (pp. 7, 45), but they occupy the same regions and provoke the same symptoms by the same mechanism, as organic diseases.

It is not strange, therefore, to see hysteria simulate brain or cord affections, since the symptoms are fundamentally the same, and arise from defects in the same substratum: "It simply comes to this, that the symptomatology of different forms of paralysis is governed in the main by the part of the nervous system which is at fault" (p. 122). In the study of these affections we must return to the essential point, the *localisation*; their functional character does not dispense us with seeking their exact cerebral or spinal seat (p. 2). We must be able to distinguish a functional defect of the cerebral cortex of the lumbar cord (p. 5). Thus shall we be able to make distinctions in this over-loaded group of functional diseases, and recognise the fact that many differ from others, and must not be designated by the vague term "hysterical."

We have endeavoured to summarise correctly the author's view on this essential point; we may now, without criticising it, indicate wherein the further study of the book leaves us still in difficulty and hesitation. The differences which Bastian allows to subsist between organic and functional diseases are after all of small import. The absence of observable lesions is not definitive, and depends on the insufficiency of our means of investigation. The duration and final cure of the disorder are not signs of any precision; hysterical symptoms may last for years and never disappear (an old Salpêtrière patient, Aurel, is hemi-anæsthetic at 75 years of age as she was at 20); whilst certain organic disorders (scleroses, compressions, peripheral neuritèdes especially, of which the author does not always seem to hold sufficient account) may be recovered from more or less completely. The third characteristic difference of intensity is not in itself of any value, and is furthermore not very exact. Hysterical symptoms, far from being always attenuated, are mostly very clear and intense. Hysteria, as Charcot said, is essentially a disease with "excessive manifestations." Its palsies, anæsthesiæ and contractures are more definite and pronounced than elsewhere. Thus the differentiæ between organic and functional diseases tend to disappear. Perhaps the two orders may still be etiologically distinguished; but clinically they will run into one another and consider their localisation only. The group of hysterical symptoms should disappear as useless. But may we accept these conclusions; and are hysterical contractures and palsies clinically undistinguishable from organic nerve symptoms? And have the words "functional troubles" lost every meaning? The expression "functional disease" appears to us as possessing some exactness even when taken in its literal sense; it is a disease of the "function" as opposed to the organ. The function is an *ensemble* of acts executed most frequently by several organs, co-ordinated so as to work out a common end. It is rare that an organ, taken singly, accomplishes a function; it mostly collaborates with other organs to fulfil a single function; and inversely it often enters as a constituent in several functions. The leg is an organ, and walking is a function; the legs are associated together and with the muscles of the trunk in carrying on the function; and on the other hand the leg comes into play in other functions, such as dancing, jumping, &c. An affection of the leg leaves the other parts intact but suppresses its action in all functions, dancing as well as walking. On the contrary, a disease which allows the patient to jump, to dance, move the legs in bed, and push them firmly against an obstacle, but which makes walking, and walking alone, impossible, is a disease of the function. Disturbances thus systematised are frequent; thus, in astasia, the patient without paralysis of a single muscle cannot stand; in abasia he falls as soon as he tries to walk normally, whilst he can skip over a rope or go on tip-toe. The same obtains in the numerous professional neuroses, in writing, sewing, &c., when the hand is fully capable to accomplish all other movements; in mutism, with loss of speech alone without any interference with the intellectual processes; still more in the loss of one's native language, with preserved use of a foreign tongue, &c. In such very definite cases the disturbance of function stands in strong contrast with disease of an organ.

But, will it be said, when the whole limb is paralysed the distinction is more difficult. No doubt, but the characteristic systematisation still exists. The paralysis is too complete, and is too sharply limited to be explained by coarse anatomical lesions. The hemiplegia affects the arm and the leg, without invading the facial area; it is a "double monoplegia" rather than a hemiplegia as Charcot frequently puts it. The arm is completely paralysed, and total anæsthesia extends upwards to the shoulder like a coat-sleeve. The eye is wholly anæsthetic, including lids and conjunctiva; and yet there is not the least implication of the facial or the leg, and the rest of the face preserves its complete sensitivity. It would seem that the function of the organ, and more especially the natural and common conception people have of this function, plays a more important part in the localisation of the symptoms than the anatomical arrangements of the limbs and nervous centres.[1]

It may be said that this very systematisation, this function of walking, writing, speaking, must have its anatomical counterpart, its unit in some single cerebral organ. It is possible; but there is absolutely no proof of the fact. The distribution of nerves and arteries, the cerebral centres, represent coarser systematisations, and determine movements of organs participating in various functions. We know in a way the centre of lip-movements; but what about the centres presiding over the articulation of a French, English, or German word? Hence the saying: "Hysteria behaves in its paralytical and other manifestations, as if anatomy did not exist."[2] Far from being explicable through our knowledge of cerebral localisations, hysteria is opposed to our *actual* knowledge of brain mechanisms.

The term "functional disease" may be of a temporary description, which will become useless when we shall have accurately localised all these complex and delicate systematisations; but until then it has a meaning and designates a set of phenomena different from those named "organic."

These remarks will find their application when we follow Bastian in his anatomical classification of "functional" disturbances. He distinguishes a certain number of paralyses, which he connects with cerebral troubles; and these he considers as hysterical properly so-called. What justifies, to his eyes, this localisation is the single—important enough—characteristic of loss of muscular sense, or kinæsthetic anæsthesia, which accompanies these paralyses. Hence the author places their seat in the Rolandic and marginal convolutions which govern the affected limbs. When these symptoms are complicated by sensory anæsthesia, a similar defect must exist, according to Bastian, in the sensory portion of the internal capsule, the portion of the corona radiata which is between the optic thalamus and the posterior portion of the lenticular nucleus (pp. 18, 121). Finally, in certain cases of hysterical mutism, or complete simple aphemia with intellect unimpaired, the author assumes a disturbance among the descending fibres from the Rolandic convolutions, and the associative fibres which connect the kinæsthetic speech centre of the third frontal sulcus with the bulbar centres of articulation (p. 50).

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[1] We have often endeavoured to put this point in evidence (see especially our lecture on "Hysterical Anæsthesia," *Arch. de Neurol.*, 1892, p. 329. See also Freud, *Ibid.*, 1893, p. 29).

[2] Freud, *loc. cit.*

We must pass over the two last localisations, the full discussion of which would lead us into the wide field of hysterical anæsthesia and mutism; a study of the first and most important will, moreover, cover some of the ground. Rey Régis ("Histoire Naturelle de l'âme," 1789) had already recognised the existence of motor paralyses, depending on loss of memory of motor force, due to certain cerebral lesions; and Charcot had also mentioned this forgetfulness of the motor images.[1]

Bastian endeavours to give precision to these ideas. He recalls his own important theories, in which he established the sensori-motor function of the Rolandic area. These centres register kinæsthetic impressions, preserve the images of the muscular sensations felt in the performing of the several movements, and by the reproduction of these images determine and guide the reproduction of the movements. Destruction of functional disturbance of these centres must give rise to three symptoms: (1) loss of muscular sense in the corresponding peripheral organ, loss of sense of position of the limb, of sense of weight, &c.; (2) loss of images related to the movements of the organ; (3) its motor paralysis. Horsley's cases and experiments show this to be the case for destructive lesions of the ascending convolutions. Bastian assumes that similar conditions obtain in hysterical paralysis; the affected limbs are paralysed as to movement and muscular sense. The author notes this curious fact,[1] that the patient cannot recall to his imagination the movements of the affected limb: "Again, when asked whether she can imagine herself playing on the piano with the left hand, she at once says 'yes,' but in regard to the right hand she gives the same negative reply as before" (p. 15). This functional inertion of the Rolandic area may be more or less complete; at a lower degree it produces a slight disturbance of motion, and may, for instance, cause the curious disorder which we have studied[2] under the name of "Syndrome de Lasègue." The patient moves normally with the eyes open, but cannot move the anæsthetic arm when they are closed (p. 27, 213). When the cerebral inertia is more intense it brings about true paralysis; thus the assumption of a functional disturbance of a localised brain area explains all the phenomena of hysterical paralysis.

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[1] *Loc. cit.*, ii. 417, iii. 464.

We confess that this explanation appears to us rather too simple to account for such delicate and complex phenomena. No doubt it contains a portion of truth, and the Rolandic kinæsthetic centres may count for something in the production of hysterical paralysis; but to explain all by defects in them, it is on the one hand to say too little, on the other to say too much.

To speak only of insufficiency of the sensori-motor centres is to say too little, for hysterical paralysis is a highly complex phenomenon of which amnesia of kinæsthetic images is only one element, and often a secondary element. That a Rolandic centre be exhausted, that may be; but why should it remain exhausted a whole year? What is it that prevents reparation and keeps up this persistent exhaustion? Why should exhaustion affect the particular organ, and follow on a certain event, whilst it does not follow on some other much graver occurrence? In reality many other phenomena accompany this one, such as associations of ideas, curious troubles of attention, which play an important part in Lasègue's "Syndrôme," and especially dreams, conscious or unconscious, fixed ideas, which play a capital part in the accident, explain its duration, its localisation and systematisation. It is not without reason that Brodie, Russell Reynolds and Charcot have spoken of paralyses "dependent on idea." Is it possible to discuss these phenomena without speaking of the mental states on which they depend? Bastian alludes too briefly to suggested paralyses; he does not notice that in these paralyses, the exhaustion of the Rolandic centres, if present, is quite secondary, and that everything depends on the suggested idea. Is there any thorough-going difference between experimentally suggested and naturally developed paralyses? If their nature be identical, as many observers have affirmed, the exhaustion of the muscular centres is in neither category the essential factor. We shall relate a curious case, the last we had the opportunity of studying under Charcot. A young man, L., notices one morning on waking up, a weakness of the right arm, which becomes more marked every succeeding morning, until it becomes an almost complete paralysis. The arm presents a complete loss of muscular and tactile sensibility; a patch of anæsthesia covers the anterior and lateral aspects of the neck; and a painful and hyperæsthetic spot is found on the left flank. Shall we say that there is only exhaustion of the foot of the second frontal convolution, and another curiously limited one in the sensory part of the internal capsule? Is it not more just and scientific to take into account the facts elicited by an examination of the mental state of the subject? This young man has dreams which he expresses aloud during natural sleep, somnambulism, and attacks provoked by pressure of the hyperæsthetic point. He is a great reader of sensational novels, and has had a dream connected with the fact that his window opens on a balcony; he dreamt that a robber entered into his room and attacked him, by squeezing his neck with one hand and pressing his left flank with the knee; whilst he himself was repelling the aggressor with his outstretched right arm. The patient reproduces the scene during his attacks, when with the right arm, *no longer paralysed,* he tries to keep off the imaginary foe. Are not the symptoms localised in the neck, in the arm, in the left flank well explained by the dream? The cutaneous sensations of the neck, the feeling and movements of the arm involved by the dream, are, as it were, absorbed by it. Since the dream persists, as we have often verified it, during the waking state, though unaware of the subject, these phenomena are removed from the cognizance of the conscious personality, and their absence determines anæsthesia and paralysis. From this simple and typical instance it will appear that it is not enough to speak of insufficiency of the Rolandic area.

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[1] *Cf.* P. Janet, "Automatisme psychologique," 1889, 347-362. "Etat mental des hystériques, v. ii., p. 117.

[2] P. Janet, "Automatisme psychologique," p. 350. "Stigmates mentaux des hystériques," 1892, p. 174.

But it is also an exaggeration, as we said before, to speak of these centres as exhausted and impotent; it is to say too much. The gravest paralysis may disappear for a time under particular circumstances. We have elsewhere shown that the symptoms may disappear during attacks, delirium, intoxication, normal sleep, and especially during somnambulism.[1] "The physical exhaustion must be very slight to disappear so suddenly during the course of the malady, and the influences capable of modifying it so, appears to be the more important factors." But there is a most important topic on which we have insisted for years.[2] We have shown that in the deepest hysterical anæsthesia and paralysis neither sensation nor movement was completely abolished. They subsist and often can be brought into evidence by simple processes; but they subsist unaware to the subject, in a sub-conscious form, without his being able to perceive them, that is, connect them with his own personality. Things take place as if the elementary psychological phenomena were as real, as numerous as in normal subjects, but could not, on account of the shrinkage of the field of consciousness, and the weakness of synthetic power, cluster into a single perception of personal consciousness. We need not insist upon the theories by which we have tried to account for very complex facts; but our experiments, verified by others (*e.g.*, James, in America; Pick, Breuer, and Freud, in Germany; Binet and Laurent in France), show that we must not hastily assume arrest, exhaustion, functional insufficiency of the cortical centres in these hysterical phenomena. We cannot, therefore, accept Bastian's localisations as sufficient to account for all the facts of cerebral hysteria.

The author now comes to a very different subject. He thinks that certain functional paralyses, apparently hysterical, are so distinct from those we have been discussing as to require a different explanation. They depend upon spinal insufficiencies. This conception of spinal diseases, analogous to hysteria, inasmuch as they are functional yet very distinct as to their real and true nature (pp. 12, 55, 119), is certainly bold and interesting. It has already been broached by other authors (P. Richer, &c.), and may contain the germ of an important nosographical classification. Hence we must examine it carefully, and weigh the reasons adduced by the author in support of the distinction between these medullary paralyses from cerebral hysteria. Bastian describes two forms, the one accompanied by contracture due to pyramidal disturbance (57), the other flabby, due to diminished activity of the anterior cornual cells, and which may even be accompanied, in advanced cases, with some atrophy (97). The author, it is true, readily admits the somewhat adventurous nature of the suggestion; and we need only dwell upon the essential point, the attribution to the spinal cord of certain paralyses admittedly functional and unexplainable by any of the known types of anatomical alteration of the cord or nerves. The author's reasons for establishing this distinction are found somewhat disseminated through his work. He often insists upon the mental peculiarities of the patients. "During the whole time of her stay in the hospital she had never manifested any undue emotional disturbance. Her mental condition has been fairly equable, and there has been no tendency to exaggerate her troubles or disabilities" (p. 65). Similar remarks occur frequently (pp. 79, 90, 101); and yet reduced to these terms, can such observations be decisive? No one to-day holds to the old legend of the ever changeful, gay or amorous hysteric; among men and women too, totally different moods have been found to exist. The apathy noted more than once (pp. 75, 102) and opposed by Bastian to the hysterical temperament, we do not know why, is quite consistent with it. But we need not insist; in order to prove anything in the matter such observations must be much more precise and full.

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[1] P. Janet, "Les accidents mentaux des hysteriques," p. 121.

[2] P. Janet, "Les actes inconscients et le dédoublement de la personnalité, *Revue philos.*, 1886, vol. ii., p. 577. "L'anesthesie systématisée" and "La dissociation des phénomènes psychologiques." *Ibid.*, 1887, vol. i., p. 449. "Les actes incomscients et la mémoire pendant le somnambulisme," *Ibid.*, vol. i., p. 238.

Bastian also mentions the absence of attacks. This is more to the point; but have those patients never had any of the numerous equivalents of attacks? The author mentions mutism (p. 92), which should be carefully investigated, and do not point to a medullary trouble. Moreover, we know that attacks are often missed, especially in cases analogous to those adduced. When a hysteric suffers from a permanent symptom, contracture or paralysis, it frequently happens that no other symptom is developed, and that the patient is absorbed in her (conscious or unconscious) idea, and becomes unsusceptible to any other.

Bastian adduces contracture as a spinal, and not cerebral, symptom; as evidently similar to that which constitutes the first degree of spastic paralysis due to lateral sclerosis. This assimilation appears strange: the rapidity and exaggeration of hysterical contracture, its character when examined unknown to the patient whilst her attention is drawn away, its extraordinary modifications under anæsthetics, in somnambulism, its close relationships with the patients' thoughts and suggestions; all make it very unlike a spinal contracture. This contracture, as we have shown elsewhere,[1] is much more intimately connected with cataleptic and paralytic phenomena of cerebral origin. We do not wish to reject Bastian's hypothesis, which may be true in some cases, and we know that hysterical contracture is one of the most variable phenomena, difficult to analyse, but we may well ask for a more thorough examination of the facts before committing ourselves to such a grave conclusion.

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[1] "Stigmates mentaux," p. 196; "Accidents mentaux," p. 109.

The author next touches upon a very delicate topic, the examination of the reflexes, of the knee-jerk in particular, from the exaggeration of which he concludes to a spinal disturbance (pp. 58, 104). This exaggeration, also mentioned by others, was recently tested by us, by an examination of all the hysterical cases in the Salpêtrière. We noted that such an investigation is extremely difficult and requires the utmost precautions; and that owing to these difficulties and the enormous influence of the least emotion, the knee-jerk varies greatly even in the same patient. Even in paraplegics it was found to be irregular, *wild* rather than regularly exaggerated as in spinal affections. But should exaggeration be proved, it is open to discussion whether the fact does not depend upon conditions other than of purely spinal origin.

Bastian also mentions the absence of anæsthesia (pp. 101, 112); but we know that this is the case with many hysterics, and that typical anæsthesia is not common. Moreover, we have elsewhere shown that these patients without sensory disturbance have somewhat peculiar mental characteristics. We shall dwell more particularly upon a very important statement: that the patients did not show any sign of disturbance of the muscular sense in the contractured limbs. It is very difficult to test kinæsthesia under such conditions, and it is not sufficient to note that the patient has, with closed eyes, a knowledge of the position given to the limbs by the contracture (p. 62). For it is precisely a characteristic feature, that such patients have a persistent idea of the position of the limb, and cannot modify this idea. But when the contracture is incomplete, the presence of kinæsthesia can be made out, and Bastian justly lays stress upon the point:—"I take the view that it is of spinal rather than of cerebral type, mainly because of the absence of any distinct impairment of muscular sense in the legs, which, with such an amount of paralysis as exists here, ought to have been marked if the paralysis had been due to a functional defect in the Rolandic convolutions in relation with leg movements in the two hemispheres" (pp. 69, 96, 107). We think, if we may be allowed to say so, that the author is perhaps right, but does not convince us because he draws his conclusions rather precipitately.

We must examine—(1) Whether a paralysis without loss of kinæsthetic sense can be of cerebral origin, (2) Whether it is of hysterical nature? The answer to the first question will be clear if we consider paralyses through suggestion. In some cases they are accompanied with muscular anæsthesia, but very often occur without any. It depends upon the suggestion, the subjects' ideas, his previous psychological state, &c. Charcot had already noted the fact.[1] Bastian says as much, but does not appear to draw the due conclusions from the phenomenon, which shows that a paralysis without loss of kinæsthetic sensibility can be of cerebral origin. Moreover, clinical evidence is in accord with this statement; many paralyses clearly dependent on idea, especially if incomplete, do not involve the muscular sense. This obtains especially in astasia-abasia, and other similar systematised derangements. The paralysis is inhibitory, not amnæsic; there is a different mechanism, though evidently cerebro-psychological. As to the second question, Bastian remarks that all functional disturbances are not hysterical, and that paralysis without anæsthesia is not of this nature (p. 121). This involves minute considerations on the nature and limits of hysteria; and without entering upon them, we should not be indisposed to accept this view. Conscious fixed ideas, these pareses without muscular anæsthesia, may without inconvenience be separated from hysteria, which will thus remain more distinct; but it is not necessary to ascribe a spinal seat to them; it is enough to distinguish their different psychical characteristics.

The author has studied a number of interesting morbid states, difficult of classification; he wishes to create for them a special group, of functional spinal diseases. He may be right, after all; and his type may eventually become very important. But though the idea may be correct, the demonstration is not convincing; and for the present we are doubtful whether the cases cannot be reduced to one of the existing classes, that of organic lesions (pressure, neuritis, &c.), or that of functional cerebral troubles (hysteria, or some other psychical disturbance).

The author's bold attempt to give an anatomical account of hysteria is perhaps a little premature. At a time when we are scarcely beginning to understand the complex psychological mechanism of these symptoms, when we hardly know the coarse localisations of the simplest mental processes, may we hope to determine, with scientific precision, the seat of disturbed attention, of dreams, of fixed ideas, of alterations of personality which play the chief part in hysteria?

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[1] *Ibid.*, iii., 393.

The volume concludes with two essays on "The Neural Processes underlying Attention and Volition" and on "The Muscular Sense: its Nature and Cortical Localisation." These valuable contributions are known to the readers of BRAIN (April, 1887, and 1892). The monograph on the kinæsthetic sense is one of the clearest and best ever published, as befits the observer, who was one of the first to dwell on the importance of kinæsthetic sensations and images, and constructed the now classical theory on the subject. It is because we have all along studied with so much interest and profit the author's work on this subject, that we have ventured to criticise freely, though respectfully, this last contribution of his pen.

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The Neuroses of Development. By T. S. CLOUSTON, M.D., F.R.C.P.E. Being the Morison Lectures for 1890. Edinburgh and London; 1 vol., 8vo, pp. 138, illustrated.

THE author presents an original and interesting picture of the various neuroses from a somewhat novel point of view. Instead of studying a neurosis, such as asthma or epilepsy, taken singly and in itself, he deals with these disorders in their mutual relationships, and in their succession in the same person at different periods of his life. Neuroses are presented as diseases of development—as troubles occurring in the nervous system at different periods of its evolution. In order to understand these accidents it is necessary to remember the various phases of this evolution in man. The author admits that the brain becomes adult, can exercise its full and normal functions at the age of 25 only; and he divides into two periods the period preceding maturity. During the first, extending from birth to about 18 or 19 years of age, the brain grows in size and weight. During the first seven years this increase is very rapid, and then becomes much slower. Some simple organs, such as the kidney, when they have reached their full growth, at once exercise their functions in a perfect manner. Not so with the more complex structures, such as the brain, in which the perfecting of the functions, after the organ has reached its full size, is very evident during a further period of about seven years. Without enlarging, the organ undergoes a developmental change, becomes more highly organised, and becomes capable of higher manifestations. This further organisation escapes our methods of investigation, and we fail to discover any appreciable differences between the brain texture of a child of 10 and that of a man of 25. That differences exist, however, is shown by the comparison of mind and conduct.

To this first division, we must add another from another point of view, though no less important; the author calls "pre-reproductive," the period antecedent to puberty, which is followed by the reproductive period with its characteristic peculiarities. Hence the brain, in the pre-maturity period, goes through several stages, marked by critical phases: crisis of birth, ages of suckling, of dentition, of fastest increase of brain weight (4 to 7), puberty, period of greatest proportional increase in general body bulk, height, and weight next to the first year of life (14 to 17), of the gradual and steady maturity and solidification of the bones and tissues generally (18 to 25), and finally that of the completion of the organism, structurally and functionally, sexually and mentally, about 25.

These critical phases through which the brain passes in the course of its development are not without their dangers, and it is then that all original debilities mostly tend to become manifest. The latter depend on hereditary taint, and which is always found among the ascendants of epileptics and choreics. The author's volume is devoted partly to the study of these striking examples of degenerescence. He expands the theories of Morel and Moreau de Tours; insisting upon the fact that hereditary influences modify chiefly the organs whose development is slowest, and that these alterations manifest themselves chiefly at the time when growth and development have reached their summit.

The author, in common with Magnan and his school, endeavours to determine what are the apparent external manifestations of degenerescence, and one lecture is devoted to the description of some of the morphological signs of a bad neurotic heredity that appear during development. In several interesting passages he shows that physical ugliness must not be considered as an entirely normal fact, but is a sign of a faulty nervous predisposition. Thus in the case of J. R., he says (p. 21): "Her trophic centres have produced a coarse skin, a bad expression, a deformed and ugly person." And on page 34 we read: "There has been a lack of harmonious progress; the bodily ideal of the age of adolescence has not been attained through the working of an evil heredity. Some features have been retarded in growth, some distorted, some overgrown." Many other corporeal defects are attributable to the same cause: asymmetry, dwarfishness, hunchback, squint, ungainly movements, horse-laughing, hobble-de-hoyism, uncomfortable limbs, malformation of ears, jaws, teeth, hands, and feet. Some of the author's statements are somewhat doubtful; at any rate, we may ask for proof that all these deformities really point to a neuropathic heredity.

The question of deformation of the palate and superior maxillary bone (ogival palate of the French) is more fully discussed. The author has made a special examination of the palate in numerous normal and abnormal subjects, and makes three classes: the normal palate, wide and not high; the neurotic palate, narrow and high (V or saddle shaped); the irregular and deformed palate. Statistics show that the latter forms are much more frequent among idiots and degenerates; and they correspond with a narrow cranial base—in other words, with a narrow brain, especially in the anterior lobes.

Persons offering such stigmata of degenerescence are subject to various nervous diseases, which manifest themselves at various periods of development. Morbid heredity is the stronger the earlier it becomes apparent, and may even disturb the embryonal functions, producing acephalous, harelip, cleft palate, spina bifida, deformed palate, and, above all, genetous idiocy. Later, during the period of greatest brain growth (up to 7 or 8 years), degenerescence is marked by rickets, night-terrors, convulsions, infantile paralysis, epilepsy, stammering, hypermetropia, convergent strabismus, lamellar cataract, choroiditis disseminata, strumous ophthalmia, ichthyosis, xeroderma pigmentosum, dermatitis herpetiformis, some variety of idiocy and imbecility, liability from neurotic causes to sudden rise of temperature, great susceptibility to attacks of all micro-organisms and zymotic poisons, night febrile delirium of children with temperatures of 99° to 101°, tubercular meningitis, Friedreich's disease, hydrocephalus, deaf-dumbness, &c. In the third period (7 to 13) of co-ordination of motion and emotion, one sees chorea, epilepsy, asthma, somnambulism, megrim, myopia, convergent strabismus, &c. Finally, during the fourth period, puberty and adolescence, the neuroses are: epilepsy, asthma, chlorosis, menstrual defects, hysteria, interstitial keratitis, megrim, adolescent insanity and stupidity, impulsiveness, dipsomania, perversion of the moral sense and volition, frothy religiosism, perverted sexual instincts, unfounded aversion to relatives, arrested body-growth, adolescent ugliness, some kinds of joint diseases, ingrowing nails, acne, subunguial exostosis, naso-pharyngeal polypi, beardlessness, barrenness, phthisis, acute rheumatism, &c.

This simple enumeration shows what a number of questions are raised in this volume. One cannot feel uneasy, however, at the number and variety of complaints attributed to one and the same cause; and one recollects the teaching—not so very long ago—that typhoid fever was a developmental disease which young people had to go through. The author somewhat neglects, among the complex causes at work in the causation of disease, the external influences. A disease is always the result of two factors—of a certain innate predisposition and of an occasional cause. In some cases, at least, the second cause is of more moment than the first. It is thus for moral as well as physical phenomena. Evidently, a hereditary predisposition is necessary for the development of obssessions or hysteria. But it is nevertheless true, that great emotions, suffering, or afflictions have determined the disease and its peculiar form. The same patients placed under different circumstances would probably not have been affected in the particular manner they have. Inversely, other persons whom we consider to be healthy, would have become hysterical or otherwise affected if placed in the position of the former. We have to deal with a very complex mixture of physical and moral influences, of acquired educational transformations, and of original predispositions; and every factor must be equally held into account in our explanations of the phenomena.

The author no doubt is perfectly conscious of all this, but has been only led to exaggerate somewhat one factor, in his anxiety to demonstrate its importance. The book is not intended to account fully for the neuroses, but to study them from a special point of view. Clouston has thus been enabled to give some very curious cases, and viewed from a highly interesting aspect—chorea, megrim, epilepsy, hysteria (justly considered as a mental disease), and adolescent insanity. He has proved the capital importance of neuropathic heredity, and the consequences which flow from this notion with reference to the interpretation and treatment of these neuroses.

P. JANET, M.D., Paris.

L'Idiotie, hérédité et dégénérescence mentale psychologie et éducation de l'Idiot. Le Dr. JULES VOISIN, Médecin de la Salpêtrière, Paris, Alcan, 1893.

THIS book consists of a number of lectures on Idiocy delivered by the author at the Salpêtrière during the first quarter of the year, 1893, and is an excellent summary of the conditions known to exist in the disease. The first lecture deals with the subject of heredity in general, and more especially with that form of morbid heredity which under the name of mental degeneration has been so well described by Morel. Dr. Voisin shows that though all causes which weaken the system, such as intemperance in the use of alcohol, tobacco, and morphia, malaria, defective nourishment, vitiated air, poverty and debauchery, produce degeneration in the human species, yet this human degeneration assumes a particular character in the heredity of nervous and mental maladies, so that the most diverse affections appear, and succeed one another in a family during a long series of generations. Mental and nervous diseases which are essentially hereditary in character occupy the principal place among causes of human degeneration, but they are transformed in passing from parents to children; the degeneration increases in a more or less rapid manner to end in the last stage of idiocy and cretinism, which produce sterility. Consanguinity is often a principal factor in producing this degeneration, and nothing shows this more clearly than the history of the Royal Family of Spain, from 1449 to 1700, in which the bastards alone of these families possessed their faculties in a sane condition. After dealing with the congenital and acquired causes, the author proceeds to discuss the pathological anatomy of idiocy. The lesions met with may be accidental or evolutional in character, and are seen in the cranio-facial osseous system, and in the cerebro-spinal apparatus. Amongst the accidental lesions are injuries to the cranium, cerebral compression, hæmorrhages in the meninges or cerebral substance, embolisms and thromboses. The various shapes of the cranium, such as the microcephalic, scaphocephalic, plagiocephalic, dolicocephalic, &c., are mentioned, and reference is made to craniectomy as practised by Mr. Lannelongue, in cases of microcephalic idiocy. The author agrees with Mr. Bourneville that the operation is of little use, as the microcephalic head is a sign of a vice of nutrition which affects the trophic functions of the nervous system. Facial asymmetry is often seen, as well as an arched condition of the palate, bad dentition, and badly-formed ears. The evolutional lesions of the cerebro-spinal axis are very numerous, and most important; they affect the whole or part of the brain, and are very variable in character. Hypertrophy of the brain is rare, but atrophy, sclerosis of parts of the substance, and hydrocephalus, are more commonly met with. True and false porencephalus are often seen in paralysed cases, the former being due to arrest of development, the latter the result of a destructive process due to a trouble of circulation supervening during intra-uterine life or coming on later. The author gives numerous definitions of idiocy, and concludes with one of his own:—"The idiot is an individual whose intellectual, sensory, and motor faculties are not developed, or are abnormally developed in a defective manner, or are arrested in their evolution before or some years after birth to a degree which they cannot overcome in consequence of various fœtal or chronic lesions of the brain." This, like other definitions however, does not give any idea of the part which etiology and pathological anatomy play in this affection; neither does it give an account of all the degrees of idiocy which one observes in practice. Various classifications are noticed, including that of the author, who divides idiocy into four categories, viz.: (1) complete, absolute idiocy, congenital or acquired; (2) incomplete idiocy, congenital or acquired, susceptible of amelioration; (3) congenital or acquired imbecility; and (4) mental debility. The symptoms and clinical aspect of idiocy are fully described, reference being made not only to the malformations of the cranium, asymmetry of the face, vaulted palate, and condition of the teeth and ears, but also to the condition of the limbs. The arms are often too long for the body, a condition similar to that found in monkeys, and the fingers may be webbed or increased in number; in addition, there is often paralysis with atrophy and athetosis. A long description is given of the conditions found in the genital organs, and we learn that puberty is not retarded in girls, but is delayed in boys. The author then proceeds to study the condition of the senses in idiots, and passes in review successively, sight, hearing, smell, taste, general sensibility and the muscular sense. With respect to sight, visual perception of objects is developed very late in infant idiots, and in some cases it is never present; the look is then always vague, and those affected in this way cannot by the sense of sight acquire exact notions on the form, relief and dimensions of objects. Tamburini, Morselli and Benedikt have observed in these cases occipital flattening coinciding almost always with these phenomena; and as the organ of vision is generally sound, and these patients do not see, or see badly; the reason is that their visual centre is insufficiently developed. This centre, according to Munk and Monakow, has its seat in the occipital lobes, and this fact explains to us the co-existence of occipital flattening and psychical blindness. Reference is made to the anatomical description of the brain and cranium given by Lavocat and Franck, who consider the cranium as a continuation of the vertebral column, its osseous pieces representing modified vertebræ, which contain four distinct segments of nervous substance, the seat of the special senses; the occipital vertebra enclosing the sense of sight, the sphenoidal that of taste and smell, the temporal that of hearing and the frontal the seat of intelligence. The senses of taste and smell remain generally in a rudimentary condition; some idiots are gluttonous, others, with a perverted taste, eat anything which comes in their way, such as paper, earth and pebbles; in these the sense is very little developed and they cannot distinguish sweet from bitter substances. A blunted or perverted sense of smell is frequent, and patients will be seen to inhale with indifference the vapour of ammonia and of eau de Cologne; it is necessary, therefore, to keep out of their way all injurious substances. As to the sense of hearing, deafness is much rarer than blindness in idiots, and when present is due either to lesion of the central organ, which is the more frequent cause, or to defect in the auditory apparatus; on the other hand, many have the musical sense very well developed. The muscular sense is deficient, and associated movements, such as walking, taking hold of an object, and dressing are with difficulty performed. Some idiots are constantly moving about, others on the other hand are quite inert, and will remain all day in their chair or bed; others again are subject to various automatic movements, and others destroy everything that comes in their way. Tactile sensation, general sensation, and organic sensations are sometimes diminished and sometimes absent. The chapter on language in idiots is a very interesting one. Language is shown to consist of a concourse of intellectual, sensorial, and motor images associated in a definite fashion. When one pronounces the word "orange," for example, an auditory image is called up, which, in its turn, raises the visual image of a round object; this visual image creates an olfactory and gustatory image, and from the whole of these images results the idea that we have of the word "orange;" if we wish to write or pronounce the word all the preceding images call up the special image of articulation for writing or for speech. Suppose, however, that one or many of these images are not awakened; in this case we shall have an incomplete, imperfect, or no idea of an object, and this is what happens in the case of idiots. The intellectual operations, which succeed one another at the moment of an auditory impression, are reviewed and arranged in three orders, according as the auditory perception is effected in a rude fashion, is differentiated, or becomes verbal. When these three forms of hearing are attacked separately, we have cortical deafness, psychical deafness, and verbal deafness. In idiots, who cannot speak, we have to distinguish aphasic motor idiots, from idiots who are aphasic from psychical deafness or verbal deafness. In the first case, the idiot cannot speak, but he comprehends what is said to him; in the second case, that of the profound idiot, he cannot comprehend or utter a single word; in the third, he cannot comprehend words, but can comprehend gestures.

Passing over the chapters on instincts, sentiments, intelligence, and will, we come to one on "idiotie myxœdémateuse" or "cachexie pachydermique," the names given by French writers to the disease which is known as sporadic cretinsim in this country. The well-known characters of this affection are described, and the author points out that it comprehends three orders of symptoms: (1) the cutaneous alterations which have given to it the name of pachydermic cachexia; (2) the arrest of intellectual development, the idiocy; and (3) the absence of the thyroid body. Even in those cases where the thyroid has been found to be hypertrophied, histological examination of it has shown that the hypertrophy of the connective tissue had caused atrophy of the proper elements of the gland, so that its function was suppressed. Reference is made to Mordret's researches, who found that in from 11 to 50 per cent. there was atrophy of the thyroid gland in idiocy, and who was of opinion that there was an intimate relation between thyroid atrophy and the degree of intellectual dulness. The author tries to explain the way in which the suppression or absence of the thyroid gland produces the disease, and on this point the experiments of Schiff and the observations of Ragowitch, of Albertoni, and Tizzoni are quoted. Dr. Voisin refers to the treatment of grafting portions of the gland, and injecting an organic liquid prepared from an extract of it, but makes no mention of the treatment now practised of administering by the mouth tabloids containing an extract of the gland.

The last chapter deals with the treatment of idiocy, and a full description is given of the physical, medical, moral, and educational treatment, now pursued in all institutions for the care and training of idiots and imbeciles. Great stress is laid on the prophylactic treatment, both as regards the parents and the patient, and the evils to be avoided by the parents and the means required for putting the patient into a healthy condition are fully pointed out. The book is well illustrated by cases which have come under the author's care.

FLETCHER BEACH.

Savill on the Electrical Changes of so-called Idiopathic Myopathy.

In the *Nouvelle Iconographie de la Salpêtrière*,[1] Dr. T. D. Savill publishes full particulars of an interesting case which corresponds in its history and clinical features with the Infantile Idiopathic Amyotrophy of Duchenne, but which, nevertheless, presents certain interesting changes in the electrical reactions of the muscles both to faradism and galvanism. Examples of this disease have generally been described as unattended by any electrical changes, and have consequently been regarded as due to inherent alteration in the muscle itself (a primitive myopathy), independent of any neuro-spinal lesion. The observation before us goes to show that this is not invariably so.

Cases of this class tend to gravitate, after visiting the different hospitals, to the metropolitan infirmaries, and Dr. Savill expresses the belief, founded on his experience at the Paddington Infirmary, that cases of so-called idiopathic or primary myopathy nearly always, at some time in their history, present electrical changes of some kind. This is contrary to the hitherto accepted view that this feature of unaltered electrical reactions is (after the age at which the symptoms first appear) the chief one which distinguishes cases of idiopathic myopathy from the larger group of progressive amyotrophic paralyses (anterior polio-myelitis). Dr. Savill further holds the view that many of the phenomena attending those cases are explicable on the assumption that there is a chronic generalised neuritis of very insidious onset and slow march. Certainly the case he describes presented, at one time, tenderness and pain along the course of the nerves and electrical changes, which would tally with this opinion. A case which tends to support this view has has also been recorded by Guinon in the *Nouvelle Iconographie*,[1] and the number of the journal which contains the one under consideration mentions others. Brissaud also publishes several cases having the same bearing,[2] and maintains that the clinical distinction hitherto drawn between Primitive Muscular Dystrophies and the Amyotrophies of spinal origin are not borne out by the facts. Dr. Savill also points out that cases of "primitive myopathy" and of amyotrophy accompanied by the usual electrical changes occur in different members of the same family;[3] that cases of pseudo-hypertrophic paralysis, which undoubtedly belong to the class of so-called myopathies, are sometimes attended by marked electrical changes;[4] and that these circumstances all tend to support the idea that the lesion of the myopathies is, like the amyotrophies of spinal origin, primarily in the nervous system; though on account of its slight and, may be, evanescent character, it has hitherto escaped detection.

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[1] May, 1894.

The patient was a girl named Susan S., admitted, on November 6, 1889, into the Paddington Infirmary, where she remained under observation for five years, and where she still drags on a weary existence.

The family history on the mother's side was healthy, but on the father's there was a history of epileptic and other fits, paralysis, and a tendency to drunkenness. An elder sister of the patient had certainly, from the clear account given by the mother, been attacked with identically the same form of amyotrophy, and another sister had shown some suspicious symptoms of it.

The patient, according to the mother's account, had never, even as a baby, been able to shut her eyes properly, but as a child she was bright and intelligent, and nothing abnormal was noticed about the muscles.

At the age of 7 she showed weakness and curving forwards of the spine, for which she was treated by lying upon the back, and about the same time it was noticed that the face was becoming expressionless, the speech altered, and that the lower lip was becoming very protuberant.

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[1] January, 1893—a case under the care of M. Brissaud in 1890.

[2] "Leçons sur les Maladie Nerveuses," Paris, 1895, p. 344, &c.

[3] See also Charcot, "Lectures on Diseases of Nervous System," New. Syd. Soc., vol. iii., p. 64, and Cenas and Douillet, *Loire Médicale*, Nos. 7 and 8, 1885.

[4] Bédard et Rémond, *Arch. Gen. de Med.*, July, 1891.

When about 8 years of age the mother was attracted by the commencing deformity of the feet. At the age of 9 the deformity and weakness of the legs was sufficient to prevent her from walking, and an operation was suggested but refused. She was never able to walk after this.

At the age of 10, though the feet were deformed, there was no general wasting obvious to the mother, and the patient was a fair height for her age. However, the weakness of the legs and face was becoming progressively worse. The involvement of the arms was so gradual that it is unknown when the deformity and wasting of these first began. It seems probable, therefore, that the atrophy started in the face as a baby, and, about the age of 7 or 8, attacked the back and legs.

Condition on Admission to the Paddington Infirmary in November, 1889, *aged* 18.—The first of the pictures shows the atrophy of arms and legs, the dropped hands, and the talipes equinus.

Figs. 2 and 3 show the same deformities, and the marked spinal lordosis in other attitudes. Fig. 2 shows very distinctly in the profile the protruding lips, which the patient was quite incapable of closing. The difficulty of supporting her accounts for the blurring of the outline of fig. 3. They need no further description. At this time there was some hyperæsthesia of the skin and some general tenderness of muscles and of the distal segments of nerve trunks.

Fig. 4 shows the maximum amount of closure of the labial and visual orifices. There was at this time an extreme degree of amyotrophy, which is described in considerable detail (*loc. cit.*).

One of the most noticeable features of this case, like others of the same kind, was the marked predominance of the atrophy of the extensors over the atrophy of the flexors, resulting in "dropped hand" and "dropped foot." These deformities were due to the tonic contraction of the remaining healthy fibres of the flexor muscles, for they could be straightened almost completely by passive movement. The power of flexing the extremities was retained to some extent, but the power of extension was almost *nil*. The extremities were always very cold and livid.

Electrical examinations, both by Dr. Savill and Dr. Kilner, showed a general *diminution of faradic contractility*. Most of the muscles required three, four, or even five times the normal strength of current to produce contraction. A notable exception to this was found in the flexors of the forearm and the flexors of the lower leg on both sides of the body, which give a reaction with about a fourth of the normal strength of current, showing thus an increased faradic irritability.

The *galvanic contractility* was also, in general, somewhat diminished, the muscles requiring two or three times the normal strength of current to produce effect. Here again a notable exception was found in the flexors of the forearm on both sides, and the flexors of the left lower leg, where the contractility was somewhat increased, and KCC approximately equalled ACC. The galvanic contractility of the flexors of the right lower leg were normal, both quantitatively and qualitatively. The galvanic contractility was also, in general, *qualitatively* altered so that KCC and ACC were too nearly alike, the one being barely double the other instead of 3½ times, the only exception being the flexors of the right lower leg, as just mentioned.

Aged 19.—When the patient was admitted, at the age of 18, there were some general tenderness of the muscles and nerve trunks, and some hyperæsthesia of the skin; but about the age of 19 she developed, in addition, attacks of severe pain, accompanied by acute tenderness along the nerve trunks. These symptoms became less the following year, but never entirely disappeared. Fibrillar tremors, such as Duchenne describes in these cases, were not observed in this patient, but the limbs were affected with constant fidgety movements, and an inability to keep in any one position, resembling athetosis — another symptom which might be regarded as pointing to an involvement of the nervous system.

Aged 23.—But little change had taken place in the patient during the five years she had been under observation. She kept to her bed, the deformities seemed to be gradually getting more pronounced, and the weakness greater. The muscles were again examined by Drs. Kilner and Savill. There was, as before, a marked general diminution of *faradic contractility*, with the exception of the flexors of the forearm on both sides, where there was a slight increase, and excepting also the flexor muscles of the left lower leg, where the faradic contractility was approximately normal.

As regards *galvanic contractility*, there was a greater general

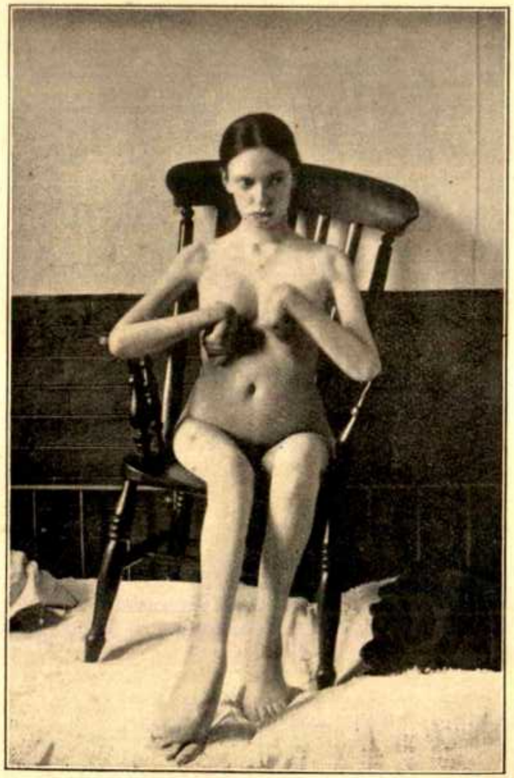

FIG. 1.

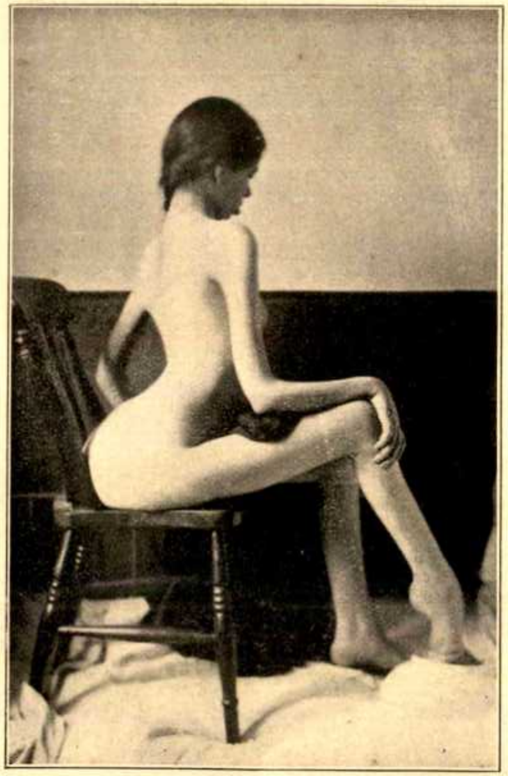

FIG. 2.

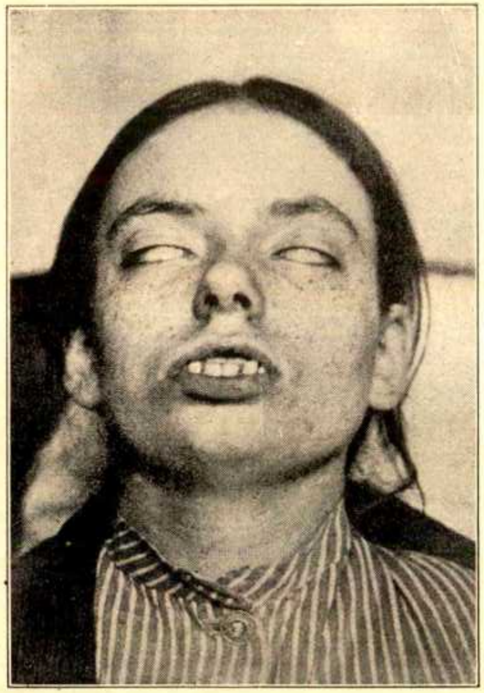

FIG. 4.

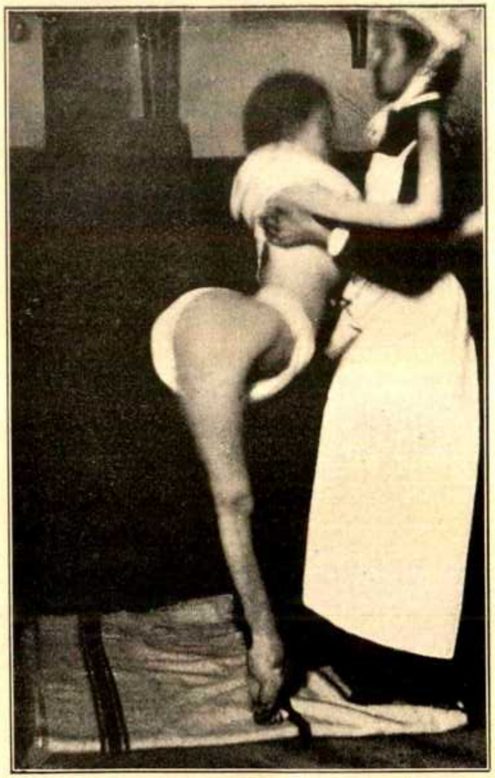

FIG. 3.

diminution than on the previous occasion, with the same exceptions as before, namely, the flexors of both forearms, and now *both* lower legs, where the contractility was fairly normal as to quantity; but KCC and ACC were now equal. Over the rest of the body KCC was only a little greater than ACC. These results are embodied in two tables which accompany the paper.

In general terms the degree of alteration of the electrical reactions was proportionate to the loss of muscular substance and power. In those muscles, however, which retained some degree of power, there was, in addition to a diminution to both currents, a qualitative alteration of the galvanic contractility. On the occasion of the second examination, the muscles which retained most power were the flexors of the legs and arms. With these exceptions the degree of atrophy was extreme.

A copious bibliography on the subject of amyotrophy is given at the end of Dr. Savill's paper. A classification of the amyotrophies is also given.

Mott and Sherrington on the Influence of Sensory Nerves upon Movement.

(Abridged from the *Proceedings of the Royal Society*, 1895, p. 481.)

(1) *Effect of Section of the whole Series of Sensory Roots belonging to the Limb.*—By the "whole series" is meant in the brachial region from the fourth cervical to the fourth thoracic inclusive; in the lumbar from the second to the tenth post-thoracic inclusive.

From the time of performance of the section onwards, as long as the animal may be kept, the movements of the hand and foot are practically abolished; the movement of grasping, which is so frequent and useful to the monkey, both with the hand and foot, never occurs at all in our experience. On the other hand, the movements at the elbow and knee, and especially the movements at the shoulder and hip, are much less impaired. The fore limb hangs from the shoulder partially flexed at the elbow; the hind limb is flexed at hip and knee. As the animal runs about it does not attempt to use the leg; the fore limb swings helplessly, with flexion at elbow and wrist and adduction at shoulder, in much the same position as if carried in a sling. The hind limb looks as if it were being held up so as to be kept off the ground while the animal runs on three legs; we are inclined to think that this appearance is deceptive, and that the position results from an equilibrium of the action of the muscles, in which purposive action on the part of the animal does not play a *rôle.* When the animal is allowed to climb a rope or the side of the cage, the fore limb swings more or less helplessly, and is not used for the climbing; similarly, the hind limb is kept more or less flexed at hip or knee, and is not used for the climbing. If the feeding-time be deferred and an animal, in which the *apæsthete* limb is an arm, be tested by offering it fruit after the sound arm has been secured behind the back, there is no attempt to use the apæsthete limb for reaching the food, but the neck is thrust forward in order for the mouth to seize it. If the fruit be placed in the hand of the apæsthete arm, the animal does not lift the hand, and appears quite unable to do so, even though encouraged. If, however, the hands of a tame normal monkey be secured behind its back, and as it lies on the floor, fruit be placed near it, the fruit is usually taken at once with the foot; but if the leg is apæsthete the fruit cannot be taken, although in one monkey the attempt used to be made. The foot was rapidly thrust toward the fruit by extension of hip and knee, but the foot missed its object widely, *i.e.*, by several inches, and the digits were not moved, though the ankle appeared to be slightly plantar-flexed. The impairment of motility in the limb ensues immediately upon completion of the section; that is to say, directly the effects of the anæsthetic have passed off sufficiently to allow requisite examination of the animal's ability to move its limbs, the above-described inability is discoverable as fully developed as at any subsequent period. We have kept the animals alive for various periods up to and over three months, and there has been no obvious change in the condition, either in the direction of improvement or the reverse. In the case of the lower limb, after two or three months, the constant position of flexion of hip and knee, on two occasions, gradually induced a change in the muscles of the thigh, which prevented hip and knee being properly extended, even by passive stretching.

As to the nature of the disturbance of motility in the limbs, one feature, namely, its peculiar topographical distribution, is salient and constant. The defect in motility increases from the attached base to the free apex of the limb; so that, for instance, while comparatively slight at the hip, it is successively greater at knee and ankle, and greatest (amounting as regards volition to absolute loss) in the digits.

In this respect it curiously closely simulates the impairment of motility ensuing upon ablation of the limb region of the cortex cerebri; but it is, in the monkey, somewhat *more severe* than the impairment following cortical ablation.

We find, however, that forcible and rapid movements, even of the fine joints at the end of the limb, can be induced in the animals by causing them to "struggle"; for instance, while recovering from ether inhalation, or while trying to free themselves on being held awkwardly, the whole limb at all its joints may exhibit movements; but even under these circumstances it is only once or twice that we have seen "grasping" movements of the digits, although sharp extension of the digits is not nearly so infrequent.

We are led from these and other considerations, which will be detailed in a fuller paper, to conclude that *associated movements in the limb* ("Mitbewegungen") *are comparatively little impaired* by loss of the sensation from the limb in which they occur; but that the independent and more delicately-adjusted movements which employ preponderantly the smaller and more individualised muscular masses of the hand and foot, and serve to move the digits, especially the hallux and the thumb—in fact, just *those movements which are represented most liberally in the limb area of the cortex, are extremely severely impaired, and, in some instances, are abolished.* We say "abolished" advisedly, because we are persuaded by our observations that, in the case of certain movements, *e.g.*, grasping movements of the hand and foot, opposition of pollex and hallux, the animal is rendered absolutely powerless to perform them, even under the strongest possible inducements. This conclusion has been gradually forced upon us. Although we are aware of the danger of introducing terms relating to consciousness into descriptions based almost solely on motor reactions, we believe that we cannot more lucidly state the condition of the animals than by saying that the volitional power for grasping with the hand, &c., had been absolutely abolished by the local loss of all forms of sensibility experimentally produced. Further, that this volitional power was lost immediately from the time of operation, and that there was not the slightest evidence of any recovery of it during the longest periods to which our observations extended (about four months).

This being so, it is natural to inquire what influence, if any, is exerted by the section of the posterior spinal nerve-roots of the limbs upon the reactions obtainable from the limb area of the cortex? That no diminution, but rather a slight increase of the excitability of the cortex, is the immediate result has been shown by one of us previously.[1] But the question remained, what will be the result when, for many weeks, the severance of the roots has led (as above shown) to disappearance from the limb of those very movements which the cortex, when experimentally excited, is especially able to produce? We have answered this, both by electrically exciting the cortex and by giving absinthe intravenously to produce epilepsy (Magnan). On exciting the cortex cerebri of the hemispheres in the appropriate regions for eliciting movements of the thumb, hallux, or digits, the responsive movements have been as easily elicited from the apæsthete limb as from the normal limb, and it has several times seemed to us rather more easily, that is to say, with a slightly less intensity of faradic current (the rate of interruption always remaining the same, $\frac{1}{30}''$).

As to the absinthe epilepsy, it always affected the apæsthete limb in a manner not distinguishably different from the normal limb. Convulsions sometimes started in the normal and desensitised limb simultaneously, sometimes a little earlier in one or the other; but no indubitable predominance or preference was shown by either limb. In a very few of our experiments (three) no movement was obtained in the apæsthete limb on excitation of the cortex; this was found to be explained subsequently by naked-eye degeneration of the pyramidal tract as revealed after hardening in Müller's fluid. This degeneration was due to injury accidentally inflicted upon the lateral column of the cord by the operation. The spinal tonus in the muscles of the apæsthete limb is undoubtedly much diminished.

These observations seem to us to point to the profound difference existing between the production of the finer movements of the limb in volition on the one hand, and by experimental stimulation of the cortex on the other. The fundamental importance of sensation for those finer movements of the limb, which are so especially well represented in the cortex of the ape, has by no authority been more forcibly emphasised than by Dr. Bastian. We think these experiments go even further than his arguments in pointing to the influence of sensation upon voluntary movement, inasmuch as they indicate that not only the cortex, but the whole sensory path from periphery to *cortex cerebri*, is in action during voluntary movement.

(2) *Effect of Section of a Single Sensory Root.*—In striking contradiction to the above-stated impairment of movement in the

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[1] C. S. Sherrington, *Phil. Trans.*, vol. clxxxiv., B, pp. 690, 691.

limb ensuing upon section of the whole series of its sensory nerve roots stands the effect of section of any one of the sensory nerve roots of the series singly and alone. In the latter case no impairment of movement at all results, or, at least, can with certainty be detected.

This is the case even when the largest and most important sensory root of the series is chosen for section; namely, in the upper limb the eighth cervical, and in the lower limb the sixth post-thoracic. (These are the nerves that supply the skin over the whole of the hand and foot respectively. It is to be remembered, however, that hand and foot respectively are each of them supplied with sensation by at least three sensory roots, the middle root covering the whole surface in each case.)

We attribute the fact that section of these large roots with their wide distribution over hand and foot produces so little appreciable effect, to the fact that the distribution of all the spinal nerves in the skin is an overlapping one. The extent of overlapping is great enough to prevent the section of any one nerve, even of the largest, producing actual anæsthesia of the skin in any part.

We further find that even if a field of absolute anæsthesia be actually produced by section for instance, of the seventh, eighth, and ninth post-thoracic roots, or, in some cases, by section of the seventh and eighth cervical and first and second thoracic roots, the impairment of movement resulting in the limb is comparatively slight. This is the more remarkable when the region deprived of sensibility includes some of the most highly sensitive parts in the limb, namely, those of the palm.

In such a case the retention of sensibility, although in an impaired degree, by the radial side of the palm, including pollex, is the cause, in our opinion, of the remarkable quantity and quality of movements still executed by the limb in spite of that impairment. In the case of section of the seventh, eighth, and ninth post-thoracic sensory roots, it was almost impossible, even on the closest examination, to detect any defect of movement whatever; the animal used both its feet apparently equally well in climbing or running, there was no clumsiness in picking up small objects (as a kernel) with the foot, and no hesitation in so doing. The chief detectable muscular difference between the limbs was that the knee-jerk was more brisk upon the operated side, and the calf and ham and back of the thigh were very deficient in sensation.

In the case of section of the sensory roots of the seventh and eighth cervical and first and second thoracic, besides the ulnar border of the arm, the hand, with the exception of the thumb and radial side of index, is also quite insensitive; and the sensitiveness of the skin of the thumb and index, where present, is below normal in degree. Nevertheless, although impairment of movement (especially clumsiness) is obvious, yet the condition is strikingly different to that obtaining after the whole series of roots has been cut through. Food is easily taken up from the hand and picked up from the floor with the thumb and index of the partially apæsthete limb, and the limb is freely used in progression and in climbing. The grasp of the partially apæsthete hand is easier, however, to detach from the cage than that of the normal hand.

We find, again, that a degree of impairment very nearly as great, indeed not obviously different from that produced by section of the sensory roots of the whole series of the nerves of the limb, is produced by section of just those sensory roots which supply the apex of the limb, *i.e.*, the hand and foot. If the whole hand or foot be completely apæsthete by the sections, then the peculiar degree of inability to move the limb, described at the outset of this communication, is obtained in (as far as we can see) its full extent. This result has its converse in the following, which we have also noted:

(3) *Effect of Section of the whole Series of Sensory Roots belonging to a Limb, with the exception of a Root that supplies the Hand or Foot.*—If the sensory roots of the whole series of the spinal nerves belonging to a limb be severed, with the single exception of, in the upper limb, the eighth cervical (distributed to the whole extent of the hand), and in the lower limb the sixth post-thoracic (distributed to the whole extent of the foot), a certain degree of impairment of movement of the limb results, which appears rather as weakness than clumsiness, but the degree of impairment is altogether quite slight. The limb is used freely for progression, for climbing, for picking up food, and bringing it to the mouth. The grasp of the partially apæsthete hand is easier to detach from the cage than that of the hand of the normal side.

The question naturally arises whether in the interruption of the paths of afferent impulses these can be so dissociated as to decide what share the muscular sense takes in the results observed? The afferent nerve fibres from muscles *per se* run, in every case in which they have been examined, in the corresponding spinal roots to the motor. It is possible, therefore, in the hand and foot to sever the sensory roots supplying the muscles while only partially interfering with those supplying other structures—the skin, sheaths of tendons, joints, &c. The converse is also true. On leaving intact the afferent fibres from muscle for the greater part, but interrupting all other afferent channels, we find that the defect of movement produced is nevertheless extreme, as, for instance, when the lowest four cervical and the first thoracic roots are severed. In the same way, when the fifth, sixth, and seventh post-thoracic roots are divided, the sole of the foot is apæsthete, and the defect in movement is extreme, although the afferent fibres from the plantar muscles remain to a large extent intact.

Conversely, when the seventh, eighth, and ninth post-thoracic roots are severed, the afferent fibres from the plantar muscles are completely interrupted, but the sole of the foot remains sensitive all over, owing to the sixth and fifth roots. In this case (and we have two instances of the kind), the defect of movement, if it exists at all, is not appreciable.

It must be remembered that other deeper structures are innervated by the same nerves as the skin, and these we do not wish to dissociate from the sensory channels still uninjured after interruption of all afferent tracts from the muscle itself.

One of our chief results is:—That afferent impulses, both from the skin and from the muscles, especially the former, as related to the palm and sole, are necessary for the carrying out of "highest level" movements. From observations on tame animals we are firmly convinced that there exists actual inability (and not only absence of volition) to perform the movements in question.[1]

Bastian on the Relations of Sensory Impressions and Sensory Centres to Voluntary Movements.

In a subsequent part of the *Transactions* Bastian criticises Mott and Sherrington's conclusions. After giving a *résumé* of his views on Kinæsthesis, he says:—"From the point of view of sensory guidance, cortically initiated movements are divisible into two categories:—(1) *Speech movements* which are now known to be produced under the guidance of co-active auditory and kinæsthetic centres; and (2) *Limb and other bodily movements*, which are equally well known to be produced under the guidance of co-active visual and kinæsthetic centres. In each case the kinæsthetic centres do not seem to act independently, but only in response to the primarily initiated activity in the auditory or in the visual centres respectively—these latter in all cases seeming to take the lead."

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[1] *Cf.* Chauveau's article on "The Sensorimotor Nerve-circuit of Muscles," BRAIN, 1891, p. 145.—ED.

Limb movements may be paralysed by organic lesions or by functional defects. The latter, again, may be either cerebral or spinal in seat. Bastian has described cerebral functional paralyses in the volume, of which a review is given in the present part of BRAIN. Of the *spinal functional defects* (also discussed in that part of review) he speaks as follows:—

"It is into this catagory that, in my opinion, the forms of paralysis fall that have been produced by Drs. Mott and Sherrington as a result of section of all the sensory roots of a limb.

"We have been long familiar with forms of paralysis due to structural lesions affecting the great ganglion cells in the anterior cornua of the cord; those, for instance, due to polio-myelitis or to focal lesions of different kinds involving the anterior cornua.

"Three years ago, moreover, I gave reasons for believing that there are cases of functional paralysis of spinal type, due to defects in these same regions of the spinal cord (examples of which were recorded), that ought to be distinctly separated from those of cerebral origin commonly known as "hysterical." We have now, as I am inclined to maintain, in the experiments of Drs. Mott and Sherrington, an experimental proof of the existence of one of these forms of functional paralysis of spinal origin.

"Instead of a lowered functional activity in cerebral kinæsthetic centres (interfering with volition), we have here a lowered functional activity in the motor centres themselves situated in the spinal cord, by reason of which their molecular activity is so altered that they are no longer capable of responding to ordinary volitional stimuli coming from the cerebral cortex. This, at least, is the explanation which I would suggest, instead of supposing, as the authors do, that volitional power itself 'has been absolutely abolished by the local loss of all forms of sensibility' in the paralysed limbs.

"As I have already stated, a local loss of all forms of sensibility caused by a lesion in the brain in the region of the internal capsule causes no such paralysis. There is, therefore, no ground for supposing that the animal's power of willing is interfered with by section of all the posterior roots coming from the limbs; while, on the other hand, all the details furnished by Drs. Mott and Sherrington are quite in harmony with the interpretation that the animal's ordinary will-power is unable to excite the spinal motor centres to action when their molecular condition has been altered by cutting off all the different stimuli proceeding to the corresponding region of the cord. The immediate result of this may be presumed to be a lowering of the habitual sub-activity of the motor centres upon which the condition of *tonus* in the muscles depends.

"The fact that the results are produced only when *all* the sensory roots are cut, and that 'the defect in motility increases from the attached base to the free apex of the limb,' so that 'the independent and more delicately adjusted movements which employ preponderantly the smaller and more individualised muscular masses of the hand and foot' are those which are most severely affected or abolished, although they may appear confirmatory of the interpretation of Drs. Mott and Sherrington, are, in fact, no less in accordance with that here given. This will be seen to be so if we bear in mind the overlapping of the fields of distribution of the sensory roots in the spinal cord (as shown by Sherrington), and the fact that the most delicate stimuli going to the smaller muscles might be expected to be those which would prove most impotent to rouse the sluggish spinal centres into activity. Again, when the authors say: 'We find, however, that forcible and rapid movements, even of the fine joints at the end of the limb, can be induced in the animals by causing them to "struggle"; for instance, while recovering from ether inhalation or while trying to free themselves on being held awkwardly, the whole limb at all its joints may exhibit movements,' we have facts quite compatible with my interpretation. Muscles may not respond to ordinary volitional stimuli, and yet may respond when the stimulus is strengthened under the influence of emotion. In a similar manner may we explain the fact that when the kinæsthetic centres corresponding with an apæsthete limb are stimulated by electricity, movements of the previously paralysed limb are produced just as easily as they are in a normal limb.[1] Such results may, as the authors say, be considered to point to the 'profound difference between the production of the fine movements of the limb in volition, on the one hand, and by experimental stimulation of the cortex, on the other.' This explanation is as valid for the one as for the other interpretation.

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[1] This fact shows that the cutting off of the afferent impressions by section of the sensory roots does not entail a lowered excitability of the kinæsthetic centres in the cortex, but rather the reverse, if we look to the really lowered activity in the spinal centres which the absence of tonus implies. In the case of complete cerebral hemianæsthesia, however, there is, over and above the absence of any such lowered activity of the spinal centres, another important difference between the conditions existing and those which obtain in the experiments of Drs. Mott and Sherrington, viz., no cutting off of cerebellar influence, so that the activity of the cortex and of the sub-cortical centres is less interfered with. These differences tend, I think, to explain the presence of paralysis with section of the posterior roots, and its absence with cerebral hemianæsthesia. (May 4, 1895.)

"The stimulation by electricity may, and probably does, involve a very different kind of stimulus from that which normally emanates from the cortex during a voluntary act, and how subtle are the differences that exist between the conditions leading to paralysis or not in different instances may be judged from the functional cases of paralysis of cerebral origin, previously referred to, in which there may be in the same person, in immediately successive periods, complete paralysis of the limb so long as the eyes are closed, and no such paralysis when the eyes are opened."

J. B. Charcot on Progressive Muscular Atrophy. (Thesis, Paris, 1895.—Abst. in *Rev. Neur.*, Sept. 15, 1895.)

Several recent observers have contested the existence of this disease as an independent type. The author's object is to refute their arguments, by showing that its evolution and lesions are peculiar to itself; and that the lesions of the white fasciculi depend upon the poliomyelitic process itself. Five personal cases are described; two with *post-mortem* examination. The fifth is one of subacute anterior spinal paralysis, which the author connects with the other type.

In the two *post-mortems* he found the usual antero-cornual lesions, together with sclerosis of the anterior fundamental fasciculus. This is seen, on a transverse section, as a comma-shaped band, encircling the anterior horn, and tailing off backwards in the lateral column, parallel with the contour of the cord. This peculiar zone of degeneration has been casually noticed by previous observers.

The author shows its importance and explains it as due to the destruction of the fibres originating in the "cellules de cordons." Its localisation is similar to that of the supplementary degeneration of amyotrophic lateral sclerosis. It is found in other cases of poliomyelitis; though it may be slight, and escape discovery when Pal's method is used; carmine shows it well.

The author concludes that there exists a Duchenne-Aran type of progressive muscular atrophy, as propounded by his father, the late Prof. Charcot. The sclerosis of the white fibres is no argument against it, since it depends upon the existence of a poliomyelitis, of which it forms a secondary degeneration.

Third International Congress of Psychology. (Munich, August 4-7, 1896.)

PROGRAMME OF WORK.

I. *Psychophysiology.*—(*a*) Anatomy and physiology of the brain and of the sense-organs (somatic basis of psychical life).

Development of nerve-centres; theory of localisation and of neurons, paths of association, and structure of the brain.

Psychical functions of the central parts; reflexes, automatism, innervation, specific energies.

(*b*) Psychophysics. Connection between physical and psychical processes; psychophysical methods; the law of Fechner. Physiology of the senses (muscular and cutaneous sensibility, audition, light-perception, audition colorée); psychical effects of certain agents (medicines)—Reaction-times—Measurement of vegetative reactions (inspiration, pulse, muscle-fatigue).

II. *Psychology of the normal individual.*—Scope, methods and resources of psychology—Observation and experiment—Psychology of sensations—Sensation and idea, memory and reproduction—Laws of association, fusion of ideas—Consciousness and unconsciousness, attention, habit, expectation, exercise—Perception of space (by sight, by touch, by the other senses); consciousness of depth-dimension, optical illusions—Perception of time.

Theory of knowledge—Imagination—Theory of feeling—Feeling and sensation—Sensual, æsthetical, ethical and logical feeling—Emotions—Laws of feeling—Theory of will—Feeling of willing and voluntary action—Expressive movements—Facts of ethics—Self-consciousness—Development of personality—Individual differences.

Hypnotism, theory of suggestion, normal sleep, dreams—Psychical automatism—Suggestion in relation to pædagogics and criminality; pædagogical psychology.

III. *Psychopathology.*—Heredity in psychopathology; statistics—Can acquired qualities be transferred by inheritance?—Psychical relations (somatic and psychic heredity), phenomena of degeneration, psychopathic inferiority (insane temperament)—Genius and degeneration; moral and social importance of heredity.

Psychology in relation to criminality and jurisprudence.

Psychopathology of the sexual sensations.

Functional nerve-disease (hysteria and epilepsy).

Alternating consciousness; psychical infection; the pathological side of hypnotism; pathological states of sleep.

Psychotherapy and suggestive treatment.

Cognate phenomena: mental suggestion, telepathy, transposition of senses; international statistics of hallucinations.

Hallucinations and illusions; imperative ideas, aphasia and similar pathological phenomena.

IV. *Comparative Psychology.*—Moral statistics.

The psychical life of the child.

The psychical functions of animals.

Ethnographical and anthropological psychology.

Comparative psychology of languages; graphology.

[Further particulars and forms of application may be obtained from Professor Sully, East Heath Road, Hampstead.]

Rules

AND

LIST OF OFFICE BEARERS
AND MEMBERS

OF THE

Neurological Society of London,

1895,

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1894.

LONDON
JOHN BALE & SONS
85-87-89, GREAT TITCHFIELD STREET, OXFORD STREET, W.
1895.

Office Bearers for the Year 1895.

President.

SIR WILLIAM H. BROADBENT, BART., M.D., F.R.C.P.

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ALEX. HILL, M.A., M.D.

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A. DE WATTEVILLE, M.D., B.Sc.

Treasurer.

CHARLES EDWARD BEEVOR, M.D., F.R.C.P.

Secretaries.

H. H. TOOTH, M.D., F.R.C.P.
F. W. MOTT, M.D., F.R.C.P.

Neurological Society of London.

REPORT OF COUNCIL.

JANUARY 17, 1895.

The Council has the pleasure to report that the Society has had a prosperous year. The number of Members is now 154

The interest in the meetings, shown by the attendances, is undiminished. The character of the communications detailed in the subjoined list sufficiently shows that the work of the Society has not lessened in value. At the Annual General Meeting on January 25th, 1894, the President, Dr. FERRIER, F.R.S., delivered the Inaugural Address on "Recent Work on the Cerebellum." The Balance in the hands of the Treasurer is again satisfactory, as will be seen by the accompanying Balance Sheet.

"Brain," the Journal of the Society under the editorship of Dr. DE WATTEVILLE, maintains its position as one of the leading Neurological periodicals of the day.

The Council deeply regrets the loss sustained by the Society by the deaths of Prof. HELMHOLTZ, an Honorary Member; Prof. BROWN-SÉQUARD, a Corresponding Member; and Prof. ROMANES, an Original Member and late Officer of the Society.

Proceedings of the Society during the year 1894.

(A complete list of the proceedings from the foundation of the Society to the year 1893 inclusive, will be found in *Brain*, vol. xvii., p. 19 of the Report.)

January 25th, Professor Ferrier, F.R.S., Inaugural address, "Recent Work on the Cerebellum and its Relations, with Remarks on the Central Connexions, and Trophic Influence of the Fifth Nerve," *Brain*, vol. xvii., p 1.

March 1st, Dr. D. Hack Tuke, "Imperative Ideas," *Brain*, vol. xvii., p. 179. Dr. Rayner, "Out-patient Treatment of Incipient Insanity."

April 19th, Dr. Sharkey, "A Case of Tumour of Pons and left Crus Cerebri," *Brain*, vol. xvii., p. 238. Dr. Colman (1) "Dry Museum Specimens of Brain by Whitwell's Carbolic Method;" (2) "Three Specimens of Tumours involving the floor of the Fourth Ventricle." Dr. Risien Russell, "Defective Development of the Central Nervous System in a Cat" (with lantern demonstration), *Brain*, vol. xviii. Dr. Shuttleworth, "The Brain of a Microcephalic Idiot." Dr. Bulloch, "Cerebral Psammona." Dr. Batten, "Sections of Spinal Cord in Early Infantile Paralysis." Dr. Tooth, "The Peripheral Nerves in Alcoholic Neuritis," *Path. Trans.*, 1894, vol. xlv., p. 1.

May 31st, Dr. Alexander Bruce, (1) "The Relation of the Lemniscus," *Brain*, vol. xvi., p. 456; (2) "The Connections of the Auditory Nerve" (with lantern demonstration)

July 12th, Dr. Head, "Disturbances of Sensation with especial Reference to the Pain of Visceral Disease, Part II., Head and Neck," *Brain*, vol. xvii., p. 339

December 7th, Dr. Hughlings Jackson and Mr. Openshaw, "Two Cases of Recovery from Trephining of Cerebral Abscess." Mr. Dean, "A Case of Cerebellar Abscess two and a-half Years after successful Operation." Mr. Ballance, "Successful Treatment of Cerebellar Abscess by Operation." Dr. Cagney (1) "A Case of Intracranial Disease;" (2) "A Case of Muscular Dystrophy with Vaso-motor Paralysis." Dr. Stephen Mackenzie, "A Case of Hemianopia with localised Muscular Atrophy." Dr. Ogilvie, "Optic Atrophy with peculiar Field." Mr. Stoddart for Dr. Bradford, (1) "Facial Hemiatrophy," (2) "A Case of Ophthalmoplegia." Dr. Beevor, "Facial Hemiatrophy" Dr. Whiting for Dr. Bastian, "A Case of Hemianæsthesia with Rigidity."

BALANCE SHEET, 1894.

NEUROLOGICAL SOCIETY.

Cr. Receipts.	£	s.	d.
To Balance in hand	154	8	10
Subscriptions for current year	147	0	0
Do. one in advance	1	0	0
Do. five for arrears	5	0	0
Share of profit on "Brain"	25	7	4
Excess on Cheques	0	3	2
	£332	19	4

Expenditure. *Dr.*	£	s.	d.
By Stationery—Pulman & Sons	7	0	9
Do. and Reprints—Bale & Sons ...	13	9	8
Illustrations—Danielsson	41	13	0
Do. Typographic Etching Co.	19	13	9
Do. Thévoz & Co.	6	10	6
Microscopes—Baker & Co.	4	13	0
Registrar of Medical Society—Hire of Rooms and Refreshments	4	3	0
Messrs. Macmillan	84	16	9
Bank Charges	0	0	9
Excess Subscription	1	0	0
Petty Cash Account	1	16	6
Royal Medico-Chirurgical Society—Hire of Rooms and Refreshments	6	13	0
Balance in hand	141	8	8
	£332	19	4

Dec. 21, 1894.

Audited and found correct,
D. HACK TUKE.
CHARLES E. BEEVOR, *Hon. Treasurer.*

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day

14.—Non-payment of the Subscription within twelve months after it is due shall be considered as equivalent to resignation.

15.—Absence of any Member residing within the Metropolitan area from all Meetings held during the year shall be considered as equivalent to resignation.

16.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

17.—Five Members of the Council shall form a quorum.

18.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

19.—The office of Vice-President shall be tenable for two years; one Vice-President being elected every year.

20.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election

21.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

22.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

23.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

24.—There shall be six ordinary meetings annually, of which the first held in each year shall be the Annual General Meeting.

25.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

26.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members

27.—The President shall have the power of inviting any person to attend and to take part in the scientific work of a meeting.

28.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a Special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

29.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

30.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by A. de Watteville, M.A., M.D., B.Sc.

HONORARY MEMBERS.

Mr. HERBERT SPENCER, London.
Professor WUNDT, Leipzig.
Professor DU BOIS REYMOND, Berlin.

CORRESPONDING MEMBERS.

Professor ERB, Leipzig.
,, GRASSET, Montpellier.
,, HITZIG, Halle.
,, JOLLY, Berlin.
,, LUCIANI, Rome.
,, OBERSTEINER, Vienna.
,, RIBOT, Paris.
,, SCHIFF, Geneva.
Dr. WEIR MITCHELL, Philadelphia.

⁂ Members are requested to communicate with the Secretaries when corrections are necessary.

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EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.
Pres., President.
V.P., Vice-President.

Tr., Treasurer.
Sec., Secretary.
C., Member of Council.

ORDINARY MEMBERS.

Elected.

1892 Alexander, Robert Reid, M.D., The Asylum, Hanwell, W.

1894 Andriezen, W. Lloyd, M D., West Riding Asylum, Wakefield.

1891 Ballance, C. A., M.S., F.R.C.S., 106, Harley Street, W.

O.M. Barlow, Thomas, M.D., F.R.C P., 10, Wimpole Street, W.

,, Bastian, H. Charlton, M D., F.R.C.P., F.R.S., 8a, Manchester Square, W. (Pres., 1892; V.P., 1891; C., 1886-9, 1893-5).

,, Beach, Fletcher, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey.

,, Beevor, C. E., M.D., F.R.C.P., 33, Harley St., W. (Tr., 1894; C. 1893.)

,, Bennett, A. Hughes, M.D., F.R C.P., 76, Wimpole Street, W. (Sec., 1886-7; C., 1888-91.)

1888 Birt, Ernest, M.D., L.R.C.P., M.R.C.S., West Riding Asylum, Wakefield.

1892 Bowlby, Anthony A., F.R.C.S., 24, Manchester Square, W.

1893 Bowman, Henry Moore, M.D., M.R C.P., 2, Museum Chambers, Bury Street, W.C.

1889 Bradford, J. R., M.D., D.Sc., M.R.C.P., F.R.S., 52, Upper Berkeley Street, W.

O.M. Bramwell, Byrom, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh. (C., 1894.)

1895 Bramwell, John Milne, M B., C.M., 2, Henrietta Street, W.

O.M. Bristowe, J. S., M.D., F.R.C.P., F.R.S., 13, Old Burlington Street, W. (Pres., 1891; V.P., 1889-90; Tr., 1886-8; C., 1892-94).

1892 Bristowe, Hubert Carpenter, M.D., L.R.C.P., M.R.C.S., County Asylum, Wells, Somerset.

O.M. Broadbent, Sir Wm. H., Bart., M.D., F.R.C.P., 84, Brook Street, W. (Pres., 1895; V.P., 1893; C. 1886-7.)

1894 Brodie, T. Gregor, M.D., L.R.C.P., Lindfield, Uxbridge Road, Surbiton.

1888 Brown, Sanger, M.D., 34, Washington Street, Chicago.

O.M. Bruce, Alex., M.D, F R.C.P.Ed., 13, Alva Street, Edinburgh. (C. 1895)

O M. Bruce, J. Mitchell, M.D., F R.C.P., 70, Harley Street, W.

„ Brunton, T. Lauder, M.D., F.R.C P., F.R.S., 10, Stratford Place, W.

1890 Bury, Judson Sykes, M.D., M R C.P., 10, St. John Street, Manchester.

O.M. Buzzard, Thomas, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890, V.P., 1888-9, C., 1886-7, 1891-5.)

1891 Cagney, James, M D., M.Ch., M.R.C.P., 93, Wimpole Street, W.

1889 Carter, Robert Brudenell, F R.C.S., 31, Harley Street, W.

1887 Cattell, J. McK., M.A., Ph.D., Columbia College, New York, U.S.A.

1891 Clarke, J. Michell, M D., M.R.C.P., 28, Pembroke Road, Clifton, Bristol.

O.M. Cobbold, C. S. W., M.D., F.R.C P.Ed., Bailbrook House, Bath.

1892 Cole, Robert Henry, M.B., L R.C P., M.R.C.S., Moorcroft, Hillingdon, Uxbridge.

1890 Colman, W. S., M.D., M.R.C.P., 22, Wimpole Street, W.

„ Corner, Harry, M.D., L R.C.P., Earlswood Asylum, Redhill, Surrey.

1894 Cuming, James, Prof., M S, F.R.C.P.I., 33, Wellington Place, Belfast.

1895 Davidson, Andrew, M.B., C.M., Winson Green Asylum, Birmingham.

1894 Dean, H. Percy, M D., F.R.C.S., 84, Wimpole Street, W.

O M. Donkin, H B., M.B., F.R C.P., 108, Harley Street, W.

„ Down, J. Langdon, M.D., F.R.C.P., 81, Harley Street, W.

„ Dreschfeld, Julius, M.D, F.R.C.P., 325, Oxford Road, Manchester.

„ Drummond, David, M.D., 6, Saville Place, Newcastle-on-Tyne.

1892 Dupuy, Eugène, M.D., 53, Avenue Montaigne, Paris.

1889 Eccles, A. Simons, M.B., M.R.C.S, 23, Hertford Street, Mayfair, W.

O.M. Edmunds, Walter, M B., F.R.C.S., 75, Lambeth Palace Road, S.E.

„ Ferrier, David, M.D, F R C.P., F.R.S, 34, Cavendish Square, W. (Pres, 1894; V.P., 1892-3; C., 1886-90-94.)

„ Foster, Michael, M.D., F.R.S., Shelford, Cambridge.

1888 Fox, Arthur, M.B., F.R.C.P.Ed., 16, Gay Street, Bath.

O.M. Fox, E. Long, M.D., F.R.C.P., Church House, Clifton, Gloucestershire.

„ Gaskell, W. H, M.D, F.R.S., The Uplands, Great Shelford, Cambs.

„ Gee, Samuel J., M D., F.R.C P, 31, Upper Brook Street, W.

„ Godlee, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1890 Goodall, E W., M.D., Eastern Hospitals, Homerton, E.

1892 Gotch, Francis, M.A., F.R.S, 11, Prince's Park, Liverpool.

1893 Grant, Dundas, M.D., F.R.C.S, 8, Upper Wimpole Street, W.

O.M. Gunn, R. Marcus, M.B., F.R.C.S., 54, Queen Anne Street, W.

1893 Halliburton, William Dobinson, M.D., F.R.C.P., F.R.S., 9, Ridgemount Gardens, W.C.

O M. Hamilton, D. J., M.B., F.R.S.E, University, Aberdeen.

1889 Handford, Henry, M.D., M.R.C.P., 14, Regent Street, Nottingham.

1891 Hawkins, Herbert Pennell, M.D., F.R.C.P., 109, Harley Street, W.

O M. Haycraft, J. B., M.B., F.R S E., The University, Cardiff.

1891 Head, Henry, M.D., C.M., L.R C.P., 6, Clarence Terrace, Regent's Park, N.W.

O.M. Hill, Alex, M.A., M.D., Downing Lodge, Cambridge. (V.P., 1895, C., 1892-94.)
" Hopkins, John, F.R.C.S., London Sick Asylum, Cleveland Street, W.
" Horsley, Victor, F.R.C.S., F.R.S., 25, Cavendish Square, W. (C., 1892-3.)
1889 Huggard, William R., M.D., M.R.C.P., Davos Platz, Switzerland.
" Humphry, Laurence, M.D., 3, Trinity Street, Cambridge.
O.M. Hutchinson, Jonathan, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.P., 1887-88; C., 1886, 1890.)
1889 Hyslop, T. B., M.D., C.M., Bethlem Royal Hospital, Lambeth, S.E.
O.M. Jackson, J. Hughlings, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886, C., 1887-92.)
" Jessop, W. H., M.B., F.R.C.S., 73, Harley Street, W.
1892 Jones, Henry Lewis, M.D., F.R.C.P., 9, Upper Wimpole Street, W.
1894 Jones, Robert, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.
" Kidd, Percy, M.D., F.R.C.P., 60, Brook Street, W.
O.M. Langley, J. N., M.A., F.R.S., Trinity College, Cambridge.
1888 Lawford, J. B., M.D., F.R.C.S., 55, Queen Anne Street, W.
O.M. Lees, D. B., M.D., F.R.C.P., 22, Weymouth Street, W.
1891 Macdonald, Peter William, M.D., C.M., The County Asylum, Dorchester, Dorset.
" Mackenzie, Hector William Gavin, M.D., F.R.C.P., 59, Welbeck Street, W.
O.M. Mackenzie, Stephen, M.D., F.R.C.P., 18, Cavendish Square, W.
1889 Macphail, S. R., M.D., Borough Asylum, Rowditch, Derby
O.M. Maguire, Robert, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893.)
" Mann, J. Dixon, M.D., F.R.C.P., 16, St. John Street, Manchester.
1894 Martin, Sidney, M.D., F.R.C.P., 10, Mansfield Street, W.
1893 Maude, Arthur, L.R.C.P., M.R.C.S., Westerham, Kent.
1891 May, W. Page, M.D., M.R.C.S., Goldburn, Eltham Road, Blackheath.
1890 Menzies, William Francis, M.D., B.Sc., M.R.C.P., County Asylum, Rainhill, Lancs.
O.M. Mercier, C., M.B., F.R.C.S., Flower House, Southend, Catford, S.E.
" Mickle, W. J., M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (C., 1895.)
" Morris, Malcolm, F.R.C.S.Ed., 8, Harley Street, W.
1888 Mott, F. W., M.D., F.R.C.P., 84, Wimpole Street, Cavendish Sq., W. (Sec., 1894.)
1894 Murray, George, M.B., M.R.C.P., 2, Saville Place, Newcastle-on-Tyne.
1889 Murray, H. Montague, M.D., F.R.C.P., 27, Savile Row, W.
O.M. Nettleship, Edward, F.R.C.S., 5, Wimpole Street, W.
" Nicolson, David, M.D., M.R.C.P.Ed., Broadmoor, Wokingham, Berks.
1888 Niermeyer, J. H. H., M.D., Amsterdam.
" Norman, Conolly, F.R.C.P.I., F.R.C.S.I., Richmond Asylum, Dublin.
1887 Ogilvie, George, M.B., M.R.C.P., 22, Welbeck Street, W.

1887 Ogilvie, Leslie, M.B., M.R.C P., 46, Welbeck Street, W.

,, Oliver, Thomas, M.D., F.R.C.P., 7, Elison Place, Newcastle-on-Tyne.

O.M. Orange, Wm., C.B., M D., F.R.C.P., 12, Lexham Gardens, Kensington, W.

,, Ord, W. M., M.D., F.R.C.P., 37, Upper Brook Street, W. (C., 1893-94.)

,, Ormerod, J. A., M.D., F.R C.P., 25, Upper Wimpole Street, W. (C., 1895)

,, Page, Herbert William, M.A., F.R.C.S., 146, Harley Street, W. (C., 1891-2.)

1895 Pinder, George, M.B., B.C., Seafield House, Ramsey, Isle of Man.

1888 Pitt, G. N., M.D., F.R C.P., 24, St. Thomas's Street, S E.

O.M. Poore, G. Vivian, M.D., F.R.C.P., 30, Wimpole Street, W.

1887 Pringle, J. J., M.B., F.R.C.P., 23, Lower Seymour Street, Portman Square, W.

O.M Purves, W. Laidlaw, M.D., 22, Stratford Place, W.

1894 Ransom, William B , M.D., M.R C.P., The Pavement, Nottingham.

1890 Rayner, Henry, M.D., M.R.C.P.Ed., 2, Harley Street, W.

O.M. Reid, E. W., M.B., University College, Dundee.

1893 Rennie, George E., M.D., 16, College Street, Sydney, N. S. Wales.

1895 Reynolds, Ernest S., M.D., M.R.C.P., 23, St. John Street, Manchester.

1892 Richards, Joseph Peeke, M.R.C.S., 6, Freeland Road, Ealing, W.

1891 Rivers, W. H. R., M.D., M.R.C.S., St. John's College, Cambridge.

1892 Rowe, Edmund Lewis, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

,, Ruffer, Marc Armand, M.D., 5, York Terrace, Regent's Park, N.W.

1889 Russell, J. S. R., M.D., C.M., M.R.C.P., 4, Queen Anne Street, W.

O.M. Sanderson, J. Burdon, M.D., F.R.C.P., F R S , Banbury Road, Oxford.

,, Savage, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square (C., 1886-90.)

1888 Savill, T. D., M.D., M.R.C.P., 12, Upper Berkeley Street, W.

O.M. Schäfer, E A., F.R.S., University College, Gower Street. (Pres., 1893 , V.P., 1892; C., 1886-90.)

,, Semon, Felix, M.D., F.R.C.P., 39, Wimpole Street, W.

,, Sharkey, S. J., M.D., F.R.C.P., 2, Portland Place, W. (C., 1889-92.)

1892 Shaw, James, M.D., M.Ch., Donard House, Kensington, Liverpool.

O.M. Sherrington, C. S., M.D., F.R.S., St. Thomas's Hospital, S.E. (C , 1892.)

1894 Shuttleworth, George E., M.D., Ancaster House, Richmond.

1895 Smith, F. J., M.D., M.R.C.P., 4, Christopher Street, Finsbury Square, E.C.

1887 Smith, R. Percy, M.D., F.R.C.P., Bethlem Royal Hospital, Lambeth.

1895 Smith, Telford, M.D., B.S., Royal Albert Asylum, Lancaster.

1892 Starling, Ernest Henry, M.D., M.R.C.P., B.S., 107, Clifton Hill, N.W.

O.M. Stewart, Sir T. Grainger, M.D., F.R.C.P.Ed., 19, Charlotte Square, Edinburgh.

,, Stirling, Wm., M.D., D.Sc , Owens College, Manchester.

1887 Suckling, C. W., M.D., M.R.C.P., 108, Newhall Street, Birmingham.
O.M. Sully, James, M.A., LL.D., 1, Portland Villas, Hampstead. (V.P., 1890-91.)
1889 Sutherland, Henry, M.D., M.R.C.P., 6, Richmond Terrace, Whitehall.
1888 Syers, Henry W., M.D., M.R.C.P., 3, Devonshire Street, Portland Place, W.
1891 Taylor, James, M.D. M.R.C.P., 34, Welbeck Street, W.
1889 Thorburn, Wm., F.R.C S., Rusholme, Manchester.
1892 Titchener, E. B., B.A , Ph.D., 72, Heustis Street, Ithaca, N.Y.
O.M. Tooth, H. H., M.D., F.R.C P., 34, Harley Street, W. (Sec., 1891.)
1892 Trevelyan, E. F., M.D , B.Sc, 40, Park Square, Leeds.
„ Tuckey, Charles Lloyd, M.D., C.M , 14, Green Street, Grosvenor Square, W.
O.M. Tuke, D. Hack, M.D , F.R.C.P., 63, Welbeck Street, W. (Tr., 1889-93; V.P., 1894 , C., 1887-8.)
1892 Tuke, J. Batty, M.D., F.R.C.P.Ed., 20, Charlotte Square, Edinburgh.
1891 Tuke, Thomas Seymour, M.B., M.R.C S., Chiswick House, Chiswick.
1891 Turner, William Aldren, M.D., M.R C.P., 13, Queen Anne Street, W.
O.M. Tweedy, John, F.R.C.S., 100, Harley Street, W.
1888 Voorthuis, J. A., M D., Medan, Deli, East Coast of Sumatra. (Communications to be addressed to M Seyffardt, Bookseller, Amsterdam.)
O.M. Waller, Augustus, M.D., F.R.S., 16, Grove End Road, N.W. (C., 1894.)
1894 Walker, A. Stodart, M.B., 13, Walker Street, Edinburgh.
O.M. Ward, J., D.Sc., Trinity College, Cambridge.
„ Warner, Francis, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.
1892 Washbourn, J. W , M.D , F.R.C.P., Guy's Hospital, S.E.
O M. Watteville, A. de, M.A , M.D., B.Sc., 30, Welbeck Street, W. (C., 1890; Sec. 1886-9; Editor of *Brain*.)
„ White, W. Hale, M D., F R.C.P., 65, Harley Street, W.
1894 Whiting, Arthur J., M.D., National Hospital, Queen Square, W.C.
1889 Wigglesworth, Joseph, M.D., M.R.C.P., County Asylum, Rainhill, Lancashire.
1894 Williamson, Richard T., M.D., M.R.C.P., 294, Oxford Road, Manchester.
O.M. Wilks, Samuel, M.D., F.R.S., 72, Grosvenor Street, W. (Pres., 1887; V.P., 1886; C., 1888-91.)
1893 Wills, Ernest, M.D., M.R.C.P., Claybury Asylum, Woodford Bridge, Essex.
„ Wood, Guy M., M.B., M.R.C.P., County Asylum, Rainhill, Lancashire.
1889 Wood, T. Outterson, M.D., M.R.C.P., 40, Margaret Street, Cavendish Square, W.
1892 Woodhead, G. Sims, M.D., F.R.C.P.Ed., Examination Hall, Victoria Embankment.